ARTERIAL HYPERTONUS, SCLEROSIS AND BLOOD-PRESSURE

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CHAPTER I

THE NORMAL STRUCTURE AND MOVEMENTS OF ARTERIES

STRUCTURE: PULSATILE MOVEMENT; TONE AND HYPERTONUS.

ALTHOUGH it is fully recognised that the nutrition of the body as a whole, and the vigour of its component parts and organs, depend upon the blood supply, clinical attention is almost wholly directed to the condition of the blood itself—to the enumeration of its corpuscles and the estimation of its hæmoglobin richness. The condition of the channels by which the blood reaches the various organs receives but scant consideration, although it is only necessary to mention the neglect to make it apparent that such neglect cannot be wise. Our purpose in these pages is to direct attention to the important part taken by the blood vessels in the production of clinical phenomena, varying in intensity from trifling indisposition to severe illness in which life is seriously and often immediately threatened. The subject is very wide, and no attempt will be made to cover the whole field. Only those conditions will be dealt with which are fundamental and specially illustrative.

As a preliminary to our investigation, it is necessary to recall the anatomical structure and nervous relations of the blood vessels.

Arteries consist of three coats: the external coat, or *tunica adventitia*, is formed of connective tissue in which there are nerves, lymphatics, vaso-vasorum, and in addition a considerable layer of elastic fibres next the media, not commonly described; the middle coat, or *tunica media*, is

formed of unstriped muscle fibres, which run transversely and thus surround the vessel; the internal coat, or *tunica intima*, is formed of a layer of polygonal endothelial cells with a fine line of sub-endothelial connective tissue underlying them. In some of the arteries there is an elastic lamina between the internal and middle coats, and another between the middle and external coats. These are known respectively as the *internal and external elastic laminæ*; they are by no means constantly present, and often only the internal one is represented. In the large arteries the middle coat is made up to a great extent of elastic fibres.

The arteries are supplied with nerves, which connect them with the vasomotor centre in the medulla.

The muscular tunic of arteries has, like muscle everywhere else, what is known as **tone** or **tonus**. This is a sustained measure of contraction of its individual fibres, which may be increased or diminished. It is commonly taught that it is regulated by nervous influences conveyed through the sympathetic, the constrictor fibres of which when stimulated lead to an increase of tone, or even to a distinct degree of abnormal arterial contraction; while the withdrawal of sympathetic action leads to lowering of normal tone, or to a definite relaxation of the arterial wall.

While the tone of arteries can be thus controlled by the sympathetic, there is the authority of Leonard Hill for the observation that it is soon restored after section of the vasomotor nerves. The tone under such circumstances is regarded by him as being then maintained by the blood-pressure. It will be subsequently shown that another factor has to be taken into consideration when tone is lowered or increased, as it is in the disorders which come under the notice of the physician.

In considering the movements of arteries, it is to be noted that they have firstly a **pulsatile movement**—probably a rhythmical response to the rhythmical flow of the blood, corresponding with ventricular systole and diastole. It is probably not merely the stretching and contraction of an elastic tube by a wave passing along its contained fluid, but the same kind of unceasing rhythmic movement the heart possesses. However that may be, it is important to divest

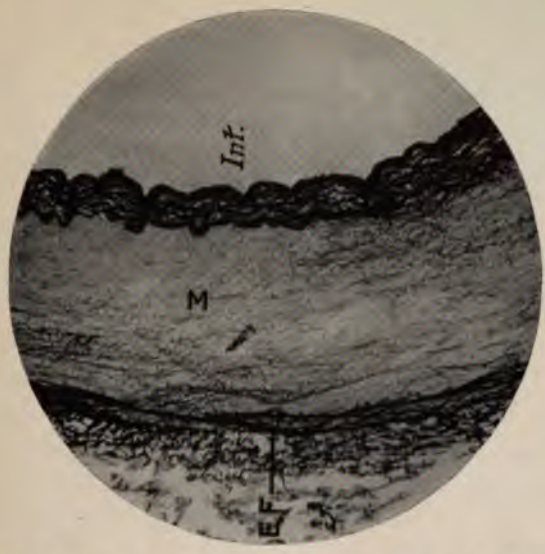

FIG. 1.—**Int.**, in the lumen of the artery, showing near it the multiplication of the internal elastic lamina; **M**, tunica media; **EF**, the layer of elastic fibres external to the tunica media.

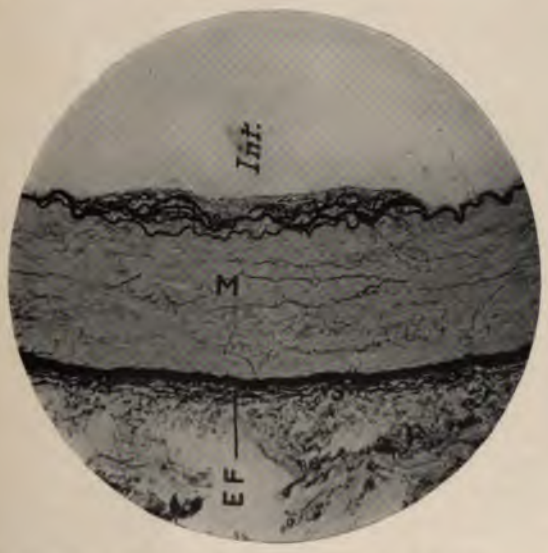

FIG. 2.—The description to Fig. 1 is applicable to this figure also.

(The sections from which these micro-photographs were taken were kindly prepared for me by Miss HUIE.)

our minds of the current notion that the arteries are a mere system of elastic tubes.

In addition to the pulsatile movement, there is a further movement, which is allied to tone, and yet is such an abnormal exaggeration of it that the terms *contraction, constriction*, and *spasm* have all been applied to it. Although occasionally recognised, it has been to a great extent ignored in clinical medicine, and its significance has, as an inevitable consequence, been overlooked. The thought of "blood-pressure" has possessed the field, to the practical exclusion of the arterial wall.

The accentuation of this normal movement is of great clinical significance, and as it varies within wide limits it is desirable to have words or terms to express the differences. The normal degree of tonicity varies, and it varies under conditions which are to be regarded as normal. It is therefore impossible to do more than have a somewhat empirical nomenclature to define its variations. What is of practical importance is to know that the tonicity does thus vary; to be able to recognise differences when they are present, and to understand that such variations are no mere chance phenomena without cause or significance. The *normal tonicity* or tone is to be noted in the soft vessel of perfect health. This tonicity is increased under physiological conditions, as during digestion and during physical effort. This is a physiological **hypertonus**; no other term expresses the fact: it is an increase of a normal state due to an increase in the intensity of normal stimuli. The term *hypertonus* has sometimes been objected to since I introduced it in this connection, but a word was required that would carry the idea which has been indicated. When the hypertonus occurs in an artery the increased measure of contraction of the muscular coat, which is necessarily implied, means that the wall of the vessel becomes somewhat thicker, that its diameter is somewhat reduced, and its lumen correspondingly diminished. The degree of contraction may exceed the limits of normal variation, and when it does so the term *hypertonic contraction*, or merely *arterial contraction*, will be used here. The word *contraction* has such a variety of meaning in medicine, and so commonly means a narrowed orifice or a

strictly limited constriction in a tube, that it requires the adjective *hypertonic* to indicate an effect which is wider and more generalised through the arterial system. The term *hypertonic contraction* will therefore be used to indicate this greater degree of arterial constriction. There is yet another condition, differing from the preceding in being a *localised constriction*. In the two preceding conditions, the "hypertonus" and the "hypertonic contraction," the contraction affects the whole systemic system. In the third condition the arterial constriction is localised, and may be so extreme in degree as to greatly diminish or completely shut off the blood flow in the affected vessels; to this condition the term *arterial spasm* is most suitably applied, and will be confined in these pages. It will from this be realised that the morbid is but an exaggeration or an intensification of the normal process. The reverse of hypertonus is loss or diminution of tone.

REFERENCE.

Leonard Hill, Schäfer's *Physiology*, 1900, vol. ii. p. 138.

CHAPTER II

BLOOD-PRESSURE AND ITS RELATION TO VESSEL CONTRACTION AND HEART POWER

DURING the past few years greatly increased interest has been taken in the important subject of blood-pressure, and in the means of clinically measuring it. It is therefore necessary to consider certain questions which have risen up around these, especially those which are of clinical importance. In no department of practical medicine is correct knowledge so essential for the satisfactory translation of mental conception into practical understanding and therapeutic application, and in this chapter I shall endeavour to make this evident.

Blood-pressure is determined by several factors: *first*, by the driving power of the left ventricle; *second*, by the channels in which the blood flows; and *third*, by the condition of the flowing blood. The second of these may be subdivided; but for the present it is important to realise that these embrace all the factors, and are the only factors which determine the measure of the blood-pressure.

The blood-pressure is, of course, the pressure exercised by the flowing blood *inside* the vessels. Physiologists measure it by means of a cannula introduced *into* a vessel, and communicating with a manometer outside it. In this way an accurate record is obtained of the pressure in the left chambers of the heart, in the great arteries, and in the great veins where they empty their blood into the right auricle. Observations of this nature are made on healthy animals.

Certain of the observations made on such a system of tubes as is provided by the vessels fall within what we may call the ordinary laws of physics; others of them are the result of the whole circuit, consisting of living structures,

being influenced and played upon both by a nervous system, and by the flowing blood itself, which is in constant contact with it.

Some of the *physical facts* are as follows. Starting from the left ventricle, the blood-pressure is highest in the aorta, and falls gradually towards the capillaries, which may be regarded as the periphery. This is true of the flow of any kind of fluid through a system of tubes. The fall in pressure, as the periphery is approached, is the result of friction between the flowing fluid and the wall of the tubes. A thick fluid flows less readily than a thin fluid; in small tubes the obstruction is greater than in large ones.

In the circulation, as it is seen in living animals, *vital processes* have to be considered, for they often determine physical changes. It is of great importance to separate these two sets of phenomena when circulatory disturbances are considered. To illustrate this, we may suppose that, as the result of *vital* influences, the vessels in a considerable area become contracted, and the circulation through them thereby impeded; the blood-pressure in the vessels above the area of constriction is raised as a *necessary consequence*. This takes place in an artificial scheme formed of rubber tubes as certainly as in the living vessels. Such a raising of pressure, if it is sufficiently great, will travel back to the aorta; if it is confined to a limited area the increased pressure will be lost before the aorta is reached, in virtue of the elasticity of the tubes. The *mean* or *average pressure* over the whole system is not affected so long as the driving power of the heart remains undisturbed; the *distribution of the pressure* is, however, altered,—where the vessels are constricted the pressure falls, where they are not constricted it is raised. The fall and the rise balance each other, and so the mean pressure is not altered. As has been said, the pressure in the aorta is often raised under the circumstances indicated, and it is this fact which has given rise to the dictum that *peripheral constriction or obstruction raises blood-pressure*. The dictum would certainly be found to occupy an honoured place in a "Proverbial Philosophy" of medicine, did such a compilation exist; but like all such dicta, it is false as well as true, and it would demand considerable ingenuity to

determine which aspect predominates. In a limited sense it is unimpeachable, yet it has had the most unfortunate results in practical medicine. It has been the common answer to many questions, and has stopped the mouths of many inquirers and seekers after truth.

Peripheral constriction, no matter how caused, raises blood pressure—but where? Is it in the constricted vessels themselves? From much that has been written the question might be answered in the positive, and yet the conception is deplorably misleading as a practical guide. The rise of pressure is behind the constriction, and the physiologists measure it in the aorta. The physical law is perfectly simple when truly applied: if the capillaries constrict, the rise in pressure is in arterioles; if the constriction includes arterioles, the raised pressure tells from the small arteries backwards, and so farther and farther back. The whole mixed conception that a constricted vessel has its blood-pressure raised has to be abandoned for a more accurate and infinitely more illuminating conception of the changes which take place.

When the raised blood-pressure reaches so far back as the aorta, what is its effect? In direct proportion to the increase of pressure in the aorta is the increase of power required by the left ventricle to open the aortic cusps and to expel its blood. This is how the raising of pressure leads to increased heart effort. If the myocardium be sound it does this in virtue of its reserve of power, which is called out through the medium of the nervous mechanism. The raising of aortic blood-pressure is the inevitable *physical* consequence of a peripheral constriction, which is a *vital* phenomenon, while the response of the heart is again a *vital* phenomenon. It is thus seen how essential it is to separate mechanical and vital factors. The response on the part of the heart is commonly represented as intended to maintain blood pressure; it seems often to be thought of as maintaining the pressure all round. If the heart did not respond there would be a fall in aortic pressure; but if it responds in proportion to the rise of pressure in the aorta, the mean is maintained, but the maintenance of the mean does not imply that an increased flow occurs in the area where the vessels are constricted. The conception that the heart is constantly battling to meet

the cry of the tissue cells for a sufficient blood supply, as a mother tends her young, is a very pretty conception, but is disproved by many morbid processes. What the heart has to cope with is the enemy at its gate—the mechanical difficulty of raised aortic pressure; and I question if the heart does more than overcome that.

The circulation as a living scheme has, however, more than one way by which the heart can be saved from the strain of such a change as has been indicated. If the systemic system be constricted the splanchnic area may dilate, and thus ease aortic pressure. The heart itself has its regulating mechanism, so that, if the aortic pressure be more than it can completely overcome, an increase of residual blood in the ventricle leads to over-distension, which so affects the vagus terminals that the heart is slowed, and the blood is given more time to pass through the constricted vessels. If the splanchnic area be dilated, as during digestion, the systemic vessels commonly become somewhat constricted, doubtless to adapt themselves to the determination of so large a volume of blood to the abdomen. This maintains aortic pressure, which might be dangerously lowered by the sudden influx of blood to the splanchnic area: to regard the condition in the systemic vessels as a raising of blood-pressure to provide the tissues supplied by the systemic vessels with sufficient pabulum. is one of the most curious of the conceptions which adorn this subject.

There is another aspect of the circulation which must be kept in view when considering the effect which peripheral changes have upon the heart, namely, this, that when the flow at the periphery is diminished, from constriction of the vessels, blood tends to accumulate on the venous side; less blood then reaches the heart, and the output of the heart, as is well known, is dependent in part on the amount which flows into it. It is equally well known that the venous system can hold all the blood in the body, that, indeed, the splanchnic vessels themselves can do so to such an extent that it is said a man can be fatally bled into his splanchnic vessels.

It has therefore to be kept clearly in mind that the vital side of the circulation provides abundant means for easing off

aortic pressure when it threatens to become excessive, and that even on *a priori* grounds the enormous increases in so-called blood-pressure which are recorded, at once ought to arouse the suspicion that there has been something wrong in the method of examination, or in the interpretation of the records obtained.

When systemic hypertonic contraction occurs, the radial arteries participate in the contraction, and this contraction is commonly regarded as "raised blood-pressure,"—an error which owes its origin, firstly, to the ignoring of the fact that the arteries are contracted; and secondly, to the conception that the rise in blood-pressure which occurs in the aorta means a rise of pressure in the radials also.

In some circumstances, which will be dealt with later, it can most reasonably be assumed that the general constriction of vessels which occurs is for the purpose of saving the tissues from an impure blood; while on the other hand, did we regard the successfully struggling heart as stimulated by the nutritional needs of the tissues, we should have to accept the existence of two antagonistic processes, maintaining an internecine strife for supremacy — the arteries exercising the right to protect the tissues, the heart heedlessly insisting on forcing through the impure and hurtful blood. And yet it seems to me as if much that has been written on the subject, say, of granular kidney, had this underlying conception for its inspiration.

The position comes to be this—that peripheral constriction raises aortic blood-pressure, and unless this can get relief by dilatation and an increased flow in another peripheral area, the heart requires increased power to overcome the increased aortic pressure. The conception of increased pressure in the constricted area, to be correct, would imply a heart effort of great magnitude, while all that the heart is really called upon to do is to empty its left ventricle contents into the aorta, the pressure in which while heightened can be eased off and relieved in the ways already referred to.

For the heart to successfully cope with increase of aortic pressure, it has the reserve of power which has been already referred to. This is well and fully recognised. The exercise of this reserve leads in favourable conditions to heart hyper-

trophy; but it has been wisely insisted upon by Broadbent and others that the very existence of hypertrophy implies that part of the reserve has already been drawn upon, and therefore that the hypertrophied heart has less reserve than the non-hypertrophied one. In practical medicine we can go further than this, for it is equally well known that the reserve of power is often very small, and may be almost absent. In some instances the response to a call for even a little extra work may be followed by an exhaustion so extreme as to be fatal.

The left heart, in such myocardial degenerations as cloudy swelling, fragmentation, fatty degeneration, acute and chronic interstitial carditis, not only possesses a low driving power, but any reserve it has is of the most flimsy character. This is shown by the embarrassment caused by the slightest movement, in extreme cases; by the disturbances of the circulation which accompany gastric digestion, or any burden thrown upon, or any disturbance of, the alimentary tract.

In the right heart not only do the changes just mentioned occur, but it is in addition the special site of fatty infiltration. This lowers the normal reserve of the right ventricle, so that any pulmonary difficulty or physical effort may suddenly overwhelm it.

In the normal sound animal, including man, increased strain calls out the reserve, and increased work is done. Every clinician, however, knows that the strain, to be successfully endured, depends upon the condition of the individual heart. It is common enough to see persons who have overtaxed their hearts, in whom the strain has exceeded the reserve, with the result that dilatation occurs, and the heart is promptly placed on a lower energy level than it previously occupied. In the first half or so of life, the period of greatest physical activity, this over-strain is rarely immediately fatal; but in the second half of life the risk is greater, and as life advances the danger becomes more urgent. This effect of age upon the circulation is universally recognised; but while this is so it is important to emphasise the fact that the myocardium differs greatly in individuals of the same age from youth onwards. This means, of course, that in different persons, at the same period of life, the heart reserve varies within wide limits, so that the general proposition that the

heart rises to the strain put upon it has in the individual at every period of life its limitations and qualifications. Seeing that the measure of the normal or sound state varies with the individual, it follows that, while the physiological principle holds, the function of the physician is to estimate the power, and the probable reserve of power, in the individual; and to recognise the effects of over-strain, however brought about. In this estimation it is necessary, indeed essential, to keep the two factors embraced in the problem always before us: the first is the work to be done, which constitutes the obstruction from the standpoint of the circulation; and, second, the power to do the work, which is synonymous with the driving power, and the reserve of power, of the ventricle, on which the strain is mainly thrown. The problem, while always presenting the same equation, requires that the value of its terms be determined in every individual instance. It is herein that both the difficulty and the interest of the physician's work lies; and herein is one illustration of how the physician, while using the physiologist's work, may branch off from him.

It has been seen how the general proposition is true, that when extra strain is thrown upon the heart it responds by extra work; and yet how that proposition has to be modified and limited by clinical observation. The extra strain upon the heart, it may be presumed, comes as a rule through changes in the vessels. Any increase of resistance in the vessels, no matter how produced, leads to extra strain on the heart. Two factors may be regarded as possibly causing increased resistance: the first is change in the condition of the circulating blood. An increase in its viscosity or specific gravity would certainly impede its passage through the capillaries; but we have on this point little or no knowledge which is of practical value, and seeing that fluid so readily passes into and out of the blood, it is very doubtful whether it will be found to be a factor of any appreciable practical importance. The second factor leading to increased resistance is an alteration in the blood channels themselves. Any narrowing of their calibre is undoubtedly a cause of resistance. That they do so narrow is also beyond question, although the importance of the fact has hitherto been obscured by the one-sided attention and devotion to the vessels as a mere system

of elastic tubes. Seeing the resistance lies in the vessels themselves, the clinical problems with which we propose to deal will be considerably simplified. In the normal state this narrowing of the vessels calls out the necessary extra work from the heart needed to overcome the increased pressure which, as we have seen, occurs in the aorta; this is the normal response of extra work to extra strain on the part of the heart. Upon our conception of what constitutes the extra work so brought out, much, it will be seen, depends. The circumstances we are at present considering are the resistance offered by a constricted set of vessels on the one hand, and a left ventricle with the ordinary amount of blood to empty itself of on the other. With these premises it follows that if the heart has sufficient reserve to do this the increased pressure in the aorta will be maintained. If, on the other hand, the heart has not the necessary reserve the ventricle is not emptied by systole; and if in any degree it fails to empty itself, the blood-pressure in the aorta is correspondingly lowered. I do not think there is any other true picture of what takes place under the circumstances with which we are dealing, and which are of common and ordinary occurrence.

We may go a step further, with apparently equal safety, and say that in certain states of the myocardium it is inconceivable that the heart can do the extra work necessary to raise the blood-pressure. With the enfeebled heart of debility, with the degenerated heart of anæmia, how is it possible to have a raised blood-pressure in the only sense in which raised blood-pressure is of practical value to the clinician?

It is necessary to lay stress upon the contention that to the clinician the all-important factor in questions of blood-pressure centres in the heart. When the vessels constrict in one region, the heart may be so powerful that the danger is vessel rupture in another; on the other hand, the heart may be so feeble that there is imminent risk of syncope. With incomplete conceptions of "peripheral resistance" and "blood-pressure" the central factor may be obscured. Whatever tends to lead away from this view of the circulation, and whatever methods tend to obscure this aspect of it require to be employed with great caution. This matter will, however, be dealt with more fully later.

CHAPTER III

DISEASES OF ARTERIES

INTRODUCTORY:
ATHEROMA—ENDARTERITIS DEFORMANS:
OBLITERATIVE ENDARTERITIS—AORTITIS:
CALCAREOUS INFILTRATION OF TUNICA MEDIA:
ARTERIO-SCLEROSIS.

INTRODUCTORY.

THE difficulty which presents itself when diseases of the arterial wall have to be considered is to be attributed to the names used to designate the conditions met with, and the confusion which exists between clinical observations, pathological teaching, and the terms used. In illustration of this it is only necessary to recall the fact that in Britain and elsewhere the term *atheroma* was used to denote perfectly definite pathological changes in vessels, while *clinically* it was commonly applied to *all* thickenings of arteries examined for clinical purposes. The artery mainly examined was, of course, the radial artery, and the condition of it came to be regarded as, in great measure, an index of the state of other arteries throughout the body. It was, however, known that the condition of the radial artery was not a certain index of the condition of the cerebral or of the coronary arteries—there was no invariable relationship—yet the relationship was sufficiently constant to warrant considerable clinical importance being laid upon changes in it. It is quite within the limits of accuracy to say that all changes in the radial artery were spoken of, and thought of, as atheroma; and that atheroma was mentally pictured as a thickening with degeneration of the arterial

wall. In fact, the conception was that of a degenerative thickening. It was further universally held that atheroma necessarily implied a *rigidity of the arterial wall*, and a loss in its elasticity. As a result of this line of thinking, based upon incomplete pathological observations, thickened arteries were regarded as atheromatous arteries, and, as atheromatous arteries were rigid arteries, therefore all thickened arteries were rigid,—such was the common reasoning.

Comparatively recently there was introduced from Germany the term *arterio-sclerosis*, which has been applied by the Germans as widely as the term atheroma has been applied in this country. They, however, distinguished between a local or nodular and a diffuse arterio-sclerosis. As etymologically the term means a hardening or induration of the arterial wall, it is applicable to nearly all forms and varieties of arterial disease. From this standpoint there is no fault to find with it, but its introduction did not help the clinical position, for the new term simply replaced the older one, without carrying any more precise pathological conception with it. It was rapidly attaining much the same position as "that blessed word Mesopotamia" had in the Highland woman's scheme of theology.

In 1901, when my first paper dealing with this subject was published, I gave a short historical summary of the position, which I may include here. Twenty years ago the term arterio-sclerosis was so unknown to British medical terminology that it was not even mentioned in the two admirable text-books published about that time respectively by Bristowe and by Fagge. Going fourteen years further back, Dr. Nöel Gueneau de Mussy wrote a historical résumé entitled *Étude Clinique sur les Indurations des Artères*; this term *induration* included atheroma. The term arterio-sclerosis is, as has been said, of German origin, and the German pathologists and clinicians certainly apply it to atheroma. Ziegler, after saying that sclerosis in an artery implies the existence of local thickening of its inner coat, proceeds to describe typical atheroma. At the same time, he did not confine the term to atheroma, for he also noted that the renal arteries and their branches in aged people are very frequently the seat of sclerotic changes, which may

simultaneously affect the arteries of other regions also, or be confined to the kidney. In this condition the intima of the arteries was notably thickened, and the lumen correspondingly narrowed or even obliterated; and he showed that this vascular change in the kidney gave rise to a special form of contraction of the organ, which he termed *arterio-sclerotic atrophy*.

Of all the German work, however, there can be no question that it was Thoma's which specially influenced opinion in this country and in America. To borrow a term from social life, he may be said to have "set the fashion" in the use and interpretation of the term in clinical medicine, and, so far as we have been able to find, pathology has not let its voice be heard beyond the lecture-room. Thoma's views may be briefly summarised as follows: the elasticity of the vessel wall becomes reduced in general diseases, acute and chronic infective diseases, long-continued disturbances of general nutrition, by many poisonous substances, and by functional overstraining of arteries from an increase of the heart's action. As a result the lumen dilates and the blood stream is retarded in the widened vessel. This retardation is the cause of a new formation of connective tissue in the intima of the widened artery. "The new formation of connective tissue in the intima renders the vessel wall more firm, so that it appears more rigid and less yielding (arterio-sclerosis, phlebo-sclerosis, angio-sclerosis)." In short, Thoma holds that the thickening of the intima is secondary to a dilatation of the vessel, and consequent on slowing of the blood stream; that the thickening is to compensate for the assumed widening, and to restore the equilibrium between lumen and contained blood which had been lost. By this means, he maintains, the rapidity of the blood flow is re-established.

If we turn to the French school we find Lancereaux in 1893 writing on "L'Endartérite ou Artério-sclérose Généralisée." He defines the conditions as a proliferation of the cells of the intima, which goes on to fatty degeneration due to failure of nutrition. Huchard mentions the fact that German authors always use the term arterio-sclerosis to signify atheroma of arteries, and he asks whether the terms are absolutely synonymous. His answer is in the negative. He

uses the term to designate a general condition of which atheroma is only one of numerous manifestations. These include visceral scleroses—all dominated by a primary chronic inflammation of the small vessels described by many authors as obliterative endarteritis (or endarteriolitis). Arteritis of the small vessels, in short, he considers the anatomical characteristic of arterio-sclerosis (p. 96).

Turning to English works of recent date, it is not necessary to go beyond the valuable *System of Medicine* which has been edited by Professor T. Clifford Allbutt. Here we look for a true representation of contemporary medicine, and in Dr F. W. Mott we find an exponent of the German view of arterio-sclerosis and an adherent to Thoma's views which have already been referred to. He places the term "arterio-sclerosis" as the title of the section, and in the introduction to the subject he says that the term "is applied rather loosely to a thickening of the vessel wall. It includes the obvious naked-eye change in the large arteries, named by some authors atheroma; by others, endarteritis deformans. It includes also arterio-capillary fibrosis, a change first described by Gull and Sutton in the walls of the small vessels, which only becomes obvious on microscopic examination." At page 320 he gives as his definition of arterio-sclerosis, "a local or general thickening of the arterial wall with loss of elasticity, occasioned mainly by fibrous overgrowth of the tunica intima, secondary and proportional to weakening of the muscular and elastic elements of the media." It seems quite clear that Dr Mott includes atheroma under the term arterio-sclerosis, and he follows Thoma in asserting that the primary change in atheroma is to be found in a degeneration of the tunica media, and that the thickening of the tunica intima is secondary and compensatory to this. He holds with Thoma that the observations made by the latter on paraffin moulds of the aorta prove this, without apparently seeing that these moulds may with equal justice be held to prove that the atrophy of the media is secondary and complementary to the hypertrophy of the intima. It is not my purpose at present to elaborate this point, but I would indeed be surprised to find that British pathologists held this to be the order of events in atheroma.

Passing from this question of the inclusion of atheroma in arterio-sclerosis, we find that Dr Mott describes a "diffuse arterio-sclerosis" (p. 329) in which the changes begin in the small arteries and capillaries, especially those of the renal cortex, brain, and heart, and are frequently associated with "nodular atheroma" of the aorta. He says that "on minute examination the muscle fibres of the media show hyaline swelling, fatty degeneration, or atrophic changes, so that the muscular elements are often not recognisable; this is especially the case in the small arteries of the kidney, where the wall of the vessel may appear to consist of a homogeneous hyaline tissue. Sometimes the degenerated atrophied fibres of the media can be made out, but nothing of the elastic lamina, the intima being thickened and represented only by a homogeneous hyaline material with but few nuclei. The result of these widespread changes is increased resistance to flow of blood through the capillaries, hypertrophy of the left ventricle, dilatation of larger arteries from degenerative changes in muscular and elastic tissues of the media, slowing of the circulation, and compensatory proliferation of the subendothelial layer of the inner coat" (p. 330). Here, again, Dr Mott strongly commits himself to Thoma's view. In America, Osler, Councilman, and others, have committed themselves to Thoma's views.

From this *résumé*, which may be taken as fairly representative of authoritative views, it is seen that the term arterio-sclerosis is applied to three conditions—(1) To atheroma; (2) to a generalised endarteritis; and (3) to a thickening of the intima, compensatory to dilatation of vessels from weakening of their middle coat.

This *résumé* could not, however, be regarded as even approximately complete without reference to various views on the relations between granular kidney and the changes in the arteries and heart which accompany granular kidney. In fact, no exposition of arterial changes could suitably ignore this side of the question.

George Johnson (1850–1873) was the first to show that thickening of the muscle coat of the arteries occurred in granular kidney, and he held that the changes in the heart and vessels were secondary to primary kidney changes. Gull

and Sutton (1872–1877) propounded the view that there occurred a generalised change or disease in the vessels, to which they gave the name, "arterio-capillary fibrosis." The changes in the kidney were regarded as secondary to this vessel fibrosis. Dickinson (1875) held that a growth of fibrous tissue in the kidney was the primary change and that the changes in the vessels were secondary to this. Rosenstein (1881) agreed with Johnson as to the thickening of the muscular coat of the arteries, but thought that the thickening of the intima was inflammatory. Ewald (1881) held that there were two forms of changes in the vessels connected with Bright's disease—first, that in which the kidney was affected primarily and was followed by hypertrophy of the heart and of the muscle coat of the arteries; and, second, that in which disease of the general vascular system was the starting-point of the kidney disease. In this latter there was arterio-capillary fibrosis, both in the kidney vessels and in the general vascular system. He thought that it was a question whether such cases should be called "Bright's disease" or not. Dreschfeld and Mahomed (1881) held practically the same view as Ewald, the latter holding in addition that there might be cardio-vascular changes without necessarily renal changes being present in all. Dr. Samuel West carried the discussion down to 1906, and discourses on the old theme as to whether the kidney lesion or the arterial change is the first to appear. He enters a protest against granular kidney being considered partly under vessels and partly under kidney.

Having thus outlined the state of opinion and belief, we may now turn to the consideration of the pathological changes occurring in arteries which are of practical importance to the clinician; and I shall deal with these in the following order:—

1. Atheroma—endarteritis deformans.
2. Obliterative endarteritis—acute aortitis.
3. Calcareous infiltration of the tunica media.
4. Arterio-sclerosis.

I adopt this order, as the first three conditions have been long known and taught in this country, while the fourth is practically a new entity, and the one which is mainly treated of in this book. I adopt and annex the

FIG. 3.—Atheromatous artery (× 15). I, the thickened and atheromatous tunica intima; M', the atrophied media corresponding to the atheromatous part; M, the hypertrophied tunica media, where there is no atheroma.

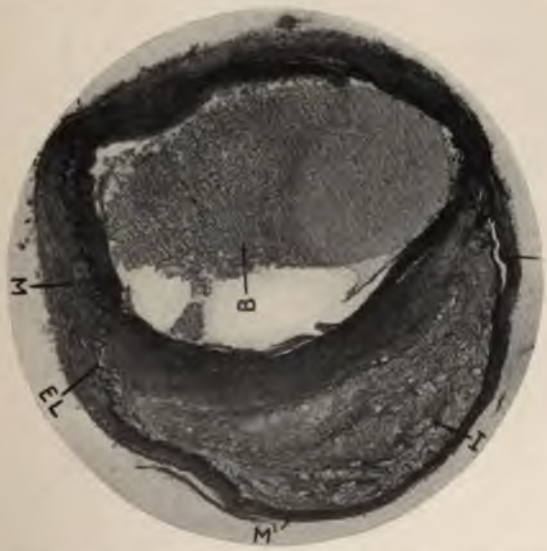

FIG. 4.—Atheromatous artery (× 16). I, thickened and atheromatous intima; M, hypertrophied media; M', atrophied media; EL, internal elastic lamina; B, recent blood clot.

term "arterio-sclerosis," as it is already in use, and as it is undesirable to multiply terms. To my mind it is quite reasonable to give a new term a limited application while declining to acquiesce in its displacing older and more definite terms.

ATHEROMA AND ENDARTERITIS DEFORMANS.

Atheroma is a focal or patchy affection of arteries. It is characterised by a local thickening and degeneration of the tunica intima. The thickening consists of a hyperplasia of the subendothelial connective tissue. It is, however, early associated with an atheromatous degeneration in parts of this thickened intima. The atheromatous change is a fatty degeneration, commonly most marked in the deeper part of the thickened tunic. The atheromatous material may become the seat of more or less calcareous deposition. At the part corresponding to these changes the tunica media is thinned and atrophied, or may even show areas of necrosis in the large arteries, as demonstrated by Cowan and others. When these changes are still further advanced and are present in the large arteries, they lead to so much deformity that the term *endarteritis deformans* was applied to them by the older pathologists. In the large arteries, in addition to the changes mentioned, there may be found atheromatous cysts, atheromatous ulcers, calcareous plates (the result of calcareous deposition in extensive atheromatous areas), and local sacculations or bulgings, the result of yielding of parts of the arterial wall before the blood-pressure following upon the atrophy or even destruction of the tunica media. The changes may be so extreme, and may so affect the arch of the aorta for instance, that it may be much dilated, a state of matters to which the term *aneurismal dilatation* is applied. Similar changes affecting a localised area of the aorta give rise to a definite aneurism.

It is however as atheroma affects smaller vessels that we are specially concerned here; so it requires somewhat more consideration. The condition is very common in the cerebral and in the coronary arteries, but is comparatively rare in the

radials. Wherever it is present it may give rise to local bulging of the vessel wall, but such bulgings do not necessarily imply a yielding of the wall. The walls at the affected points are usually much thickened and the lumen is often greatly encroached upon, lessening in a very important degree the blood channel. The thickening never uniformly affects the whole circumference of the vessel; it is always asymmetrical; it only affects small areas; it never uniformly affects long stretches of an artery, although there may be many areas closely placed to one another. It thus differs entirely from the condition to be described as arterio-sclerosis.

Etiology of Atheroma.—Atheroma is thus a focal affection of the arterial wall, affecting separate and limited areas. The changes indicate, not only points or areas of irritation, but an irritation associated with degeneration, and it is this association which characterises atheroma. The combination is what we expect to find produced by a micro-organismal cause. The theory that the degeneration is the result of impaired nutrition, due to obliteration of vasa vasorum, is quite untenable, for the following reasons, namely, that the degeneration often appears early when there is but slight thickening of the intima; the vasa vasorum may not be obliterated; and in obliterative endarteritis no such degeneration occurs, although the intima may be much thicker and the vasa vasorum be markedly affected. Dr. John Cowan of Glasgow has made some interesting researches, from which he contends that atheroma may apparently be due to a number of conditions in which micro-organismal infection of the arterial wall is probable. Now that attention has been directed to this subject, I may say that I think it probable that atheroma may ultimately be found to belong to the enlarging series of affections having as their immediate cause a micro-organismal implantation.

The typical and classical changes present in atheroma are seen in Figs. 3 and 4.

In Figs. 5, 6, and 7, atheroma gone on to advanced calcareous infiltration is shown, the figures being taken from sections of radial arteries which to the finger were recognised as hard, rigid, and calcareous during life. In all these arteries a certain amount of fairly sound muscular tissue has persisted,

FIG. 5.—Atheromatous and calcareous radial artery (× 15). **C′**, atheromatous and calcareous; **C**, media calcareous; **M**, hypertrophied media; **A**, tunica adventitia.

FIG. 6.—Calcareous radial artery (× 15). **C**, the calcareous tunica intima; **M**, portions of thick tunica media.

while in Fig. 5 it should be noted that nearly one-half of the circumference of the artery shows, not only an undegenerate, but a hypertrophied muscular coat. This anatomical fact is of clinical importance, as will be shown in a later chapter.

OBLITERATIVE ENDARTERITIS AND ACUTE AORTITIS.

The description of obliterative endarteritis, as it occurs in smaller arteries, is usually taken from the syphilitic affection found in the brain. The affection there is usually nodular, in the sense that it affects a limited bit of vessel, leading to such thickening that a considerable node or nodule is formed. The minute change is a thickening of the tunica intima, the thickening being the result of a hyperplasia of its cells. It differs, however, from the intimal thickening in atheroma in involving the intima somewhat uniformly, right round the vessel, and in showing no degeneration. The hyperplasia which leads to the thickening is progressive, and in its progress encroaches upon the lumen of the vessel, which is reduced to a minute size, or is entirely occluded. When an artery is examined in the acute stage a considerable amount of small-celled infiltration is present in all three coats. These changes in cerebral vessels are regarded as always syphilitic. Corresponding anatomical changes occur in the arteries of the kidney in arterio-sclerotic atrophy, and in chronic interstitial nephritis; they are also met with in the lungs in fibrosis, and in the neighbourhood of scirrhus cancer. In these latter a small-celled infiltration of the coats of the vessel does not occur, but the thickening of the intima goes on to complete obliteration of the vessel. A corresponding anatomical change also occurs in the smaller nutrient arteries in the brain. In none of these—especially in the lungs, kidneys, and of course in cancer—is the change regarded as of syphilitic origin. I shall again refer to these changes, as they are seen in the kidneys, under Arterio-Sclerosis.

In the aorta an acute aortitis occurs, giving rise to grey, raised, almost gelatinous-like areas, which on minute examination show hyperplastic thickening of the tunica intima

and areas of small-celled infiltration in all three coats. This condition, which anatomically entirely corresponds with the obliterative endarteritis of the smaller syphilitic arteries, I have always regarded as of syphilitic origin.

The condition, as it occurs in the cerebral arteries, is represented in Fig. 8.

CALCAREOUS INFILTRATION OF THE TUNICA MEDIA.

Clinically, calcareous infiltration of the tunica media is indistinguishable from calcareous infiltration of the thickened intima in atheroma. In fact, in advanced calcareous infiltration, it is by no means always easy to determine how much of it is intimal and how much medial. This is owing to the fact that when it is intimal there is so little of the media left that the doubt arises whether it is not the media itself which is infiltrated. The position of the elastic lamina often effectively helps in this determination. The change is one which does not require fuller consideration here. In Figs. 5, 6, and 7, the media is more or less involved in the calcareous infiltration.

ARTERIO-SCLEROSIS.

In 1901, in a paper read before the Edinburgh Medico-Chirurgical Society, I submitted the results of the examination of the radial and other arteries, taken from sixteen cases which had been under my care, and in which there had been marked thickening of the radial arteries during life. Since that time I have examined many additional cases, but they have only confirmed my previous observations, that the changes were totally different from those in atheroma, and strengthened the opinion I then expressed, that as the term arterio-sclerosis was in common use it ought to be retained, but its application confined to the changes which I then showed to be present.

The changes may be defined roughly, as great thickening of the wall of the artery, with diminution in the size of its lumen.

The changes which led to the thickening, when examined in detail, were seen to consist of—(1) a marked thickening of

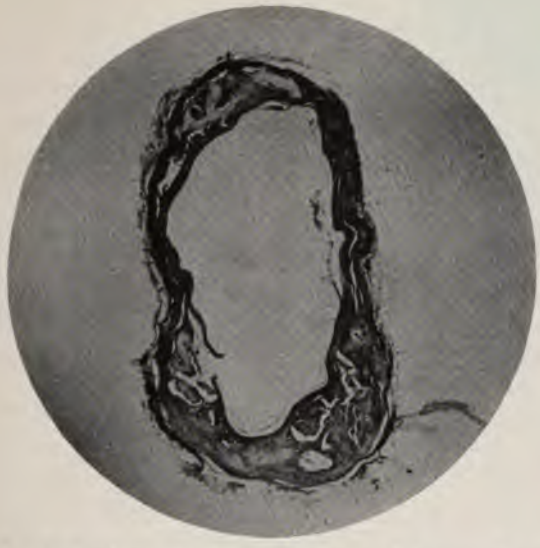

FIG. 7.—Very calcareous radial artery (× 15). Only a small portion of the right and upper part of the wall not involved.

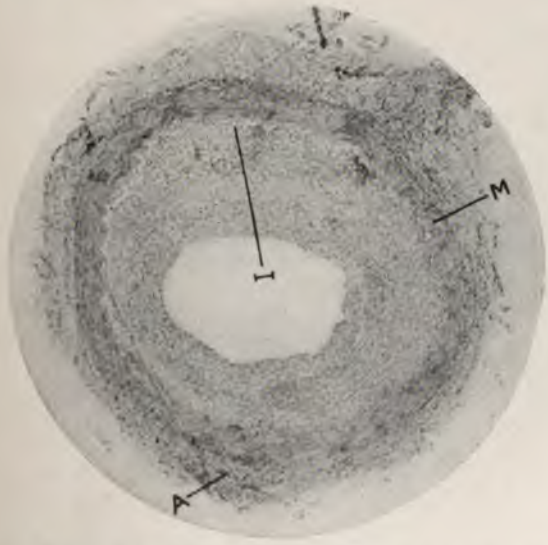

FIG. 8.—Obliterative endarteritis of syphilitic origin (× 16). **A**, tunica adventitia; **M**, tunica media; **I**, greatly thickened tunica intima.

the tunica media, due to a hypertrophy of its muscle fibres; (2) a thickening of the tunica intima, due to a hyperplasia of its subendothelial connective tissue, without atheromatous degeneration; (3) in some instances fibrous hyperplasia and thickening of the tunica adventitia.

The muscular coat might show some degeneration, but the prevalent notion that in such thickened vessels the muscle of the middle coat was replaced by fibrous tissue, and so the seat of fibroid degeneration, was found to be entirely erroneous.

These changes were not confined to limited areas of vessel wall, as in atheroma, but affected uniformly the whole length of the radial. They were found to be distributed throughout the body, the coronary and renal arteries, for instance, showing corresponding changes. The changes are shown in Figs. 9, 10, 11, 12, and 13. In Fig. 9 great thickening of the artery is shown, and is seen to be due to hypertrophy of the tunica media and some hyperplasia of the tunica intima. Fig. 10, a section of the kidney from the same case, shows, in the centre of the field, an artery with a much diminished lumen due to fibrous hyperplasia of the tunica intima, while the tunica media is atrophied; at *a* there is an occluded arteriole,

Fig. 11 shows a small thickened radial artery, which was persistently small and hard during life. The thickening here is partly a hypertonic contraction, and in part, I think, the result of a hyperplasia of the adventitia. Fig 12, a section of the kidney from the same case, shows two arteries with their lumen encroached upon by hyperplasia of the tunica intima, while the tunica media is atrophied. Fig. 13 is a section of the trunk of one of the coronary arteries, showing hypertrophy of the media and hyperplasia of the intima. Here, as is sometimes found, there is also some atheromatous degeneration in the intima.

The changes were often present when there was practically no atheroma in the aorta or the large arteries. They were, however, sometimes associated with atheroma of the aorta; while atheroma of the cerebral arteries is so common in later life, that it, so far as my observations go, often accompanies arterio-sclerosis elsewhere.

The changes present in the kidneys are of great interest, for they seem to me to explain the confusion which still exists in relation to vessel and kidney changes in "granular kidney." The changes in the renal artery *outside* the kidney are as I have already described; while the changes in its branches *inside* the kidney show a very important modification of, or divergence from, that description. I found that, inside the kidney, the arteries showed very marked fibrous hyperplasia of their internal coat, going on to complete occlusion of the vessel; that the muscular coat as a rule atrophied and might practically disappear as the tunica intima thickened; and that the external coat seemed to become denser from a proliferation of its fibrous tissue. As a result of these changes the transverse section of an artery, when occlusion was complete, showed like a fibrous globe, which might be indistinguishable from a fibroid glomerulus. The fibrous tissue might be of the hyaline variety with few nuclei; and the change has consequently been sometimes referred to as hyaline degeneration. A corresponding change occurs in the nutrient arteries of the brain, and possibly in other organs, which I have not, however, examined. Were this intimal thickening compensatory, in the sense that Thoma and his followers apply the term, it would cease with the establishment of equilibrium. This, however, does not happen, the change going on to vessel occlusion.

The changes in the kidney vessels have been much studied, and I have given a summary of the leading views propounded on the relations between the renal and vessel changes. It appears to me to be clear that error arose by applying the changes found in the arteries *inside* the kidney to all the arteries in the body. The first observer who recognised the true nature of the change in the tunica media of the systemic arteries was Dr. George Johnstone, who named it muscular hypertrophy, the reverse of the condition which occurred in the kidney; while Gull and Sutton, looking at the intimal changes and at the kidney changes together, introduced the term *arterio-capillary fibrosis*. These observers made the mistake of applying their observations on the kidney to the vessels generally. The result was, that in time the idea of a diffuse arterio-capillary fibrosis

FIG. 9.– Showing great thickening of radial artery.

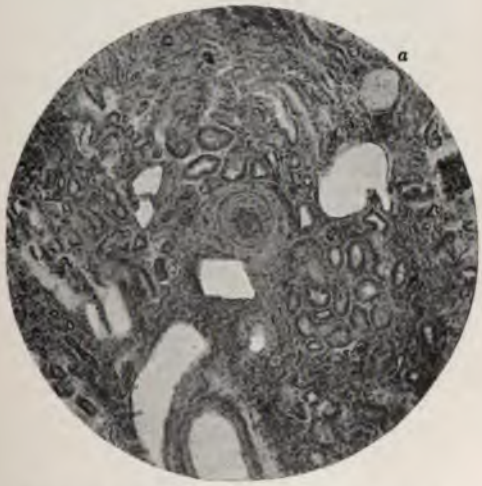

FIG. 10.—Kidney from same case as Fig. 9, showing, in centre of field, artery with greatly thickened intima and atrophied media. At *a*, occluded arteriole (× 50 diameters).

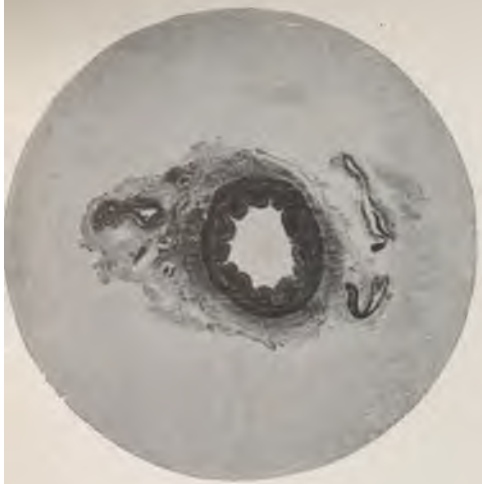

FIG. 11.—Small contracted and thickened radial artery.

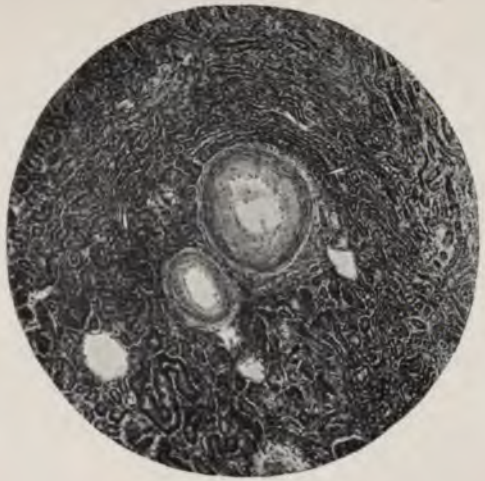

FIG. 12.—Kidney from same case as Fig. 11, showing sections of two arteries with much thickened intima and atrophy of media (×50).

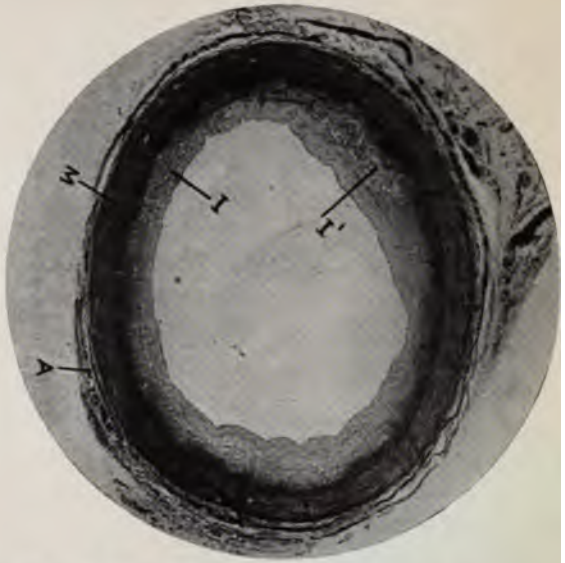

FIG. 13.—Large coronary artery from same case as Figs. 10 and 11. **A**, tunica adventitia; **M**, tunica media; **I**, thickened intima at ′, showing atheroma.

so dominated the medical imagination, that the thick arteries of chronic kidney disease were, as has already been mentioned, thought of as fibroid, if not as atheromatous; and if fibroid of course hard and rigid! The logic was sound, but unfortunately for truth the premises were wrong. To this sound logic with the faulty premises might, I think, be traced not a few of the prevailing misconceptions regarding circulatory phenomena; but, attractive as this question is, I do not propose entering upon it here.

It is necessary to revert to Johnstone's observations on hypertrophy of the muscular coat, for Savill in 1897 revived this on the strength of his own observations, and called the condition *hypermyotrophy*. In a later communication he still more fully dealt with the importance of the muscular coat of arteries; but the profession has shown no sign that it attached any practical significance to those observations or to the argument based upon them. In Savill's observations there appears not to have been the thickening of the tunica intima which was usual in my cases. In only two of my first sixteen cases was there a pure hypermyotrophy in the radial artery. This point has significance, for such arteries are clinically indistinguishable from those which have the intima thickened also; and as the two conditions have corresponding causes I include both under the designation of arterio-sclerosis.

CONCLUSION.

The term **Arterio-sclerosis** would thus be applied clinically to all thickened vessels, other than those thickened by atheromatous degeneration, and would include—(*a*) pure hypermyotrophy, (*b*) hypermyotrophy with thickening of the internal coat, and (*c*) those in which the adventitia was also thickened. For clinical purposes what is required is the recognition of diffuse permanent thickening which is not atheroma.

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CHAPTER IV

HYPERTONUS AND ITS CLINICAL RECOGNITION

(*a*) IN NON-THICKENED ARTERIES;
(*b*) IN SCLEROSED ARTERIES;
(*c*) IN ATHEROMATOUS ARTERIES.

HYPERTONUS IN NON-THICKENED ARTERIES.

A hypertonic vessel, as has been already shown, is a vessel the muscular coat of which is unduly contracted. As a result of the contraction its diameter is reduced, its wall is thicker, and its lumen is smaller. The vessel to the finger feels thicker than the perfectly normal artery, which is soft, thin-walled, and compressible. The degree of hardness varies of course with the amount of contraction. This increased thickness is very commonly mistaken for "sclerosis" or "atheroma," terms the significance of which has been already discussed. The thickening or hardness is commonly a uniform thickness, which can be felt in the radial arteries as far as they can be followed up the forearm. While this is the common character of a hypertonic artery, as revealed to the sense of touch, it has not always this characteristic. I have frequently noted that an artery, which turns out to be only hypertonically contracted, may feel as if it were made up of a series of thickened rings, or plates, or segments. An artery with these characters will almost certainly be regarded as atheromatous; and it is only by knowledge and experience that one is prevented from giving a premature opinion, which may be totally wrong. I have observed these rings and plates to disappear rapidly under treatment directed to relieve vessel contraction. Thickened arteries, especially if they be

considerably thickened, as they may be, have come to be looked upon as having undergone permanent structural thickening, for which nothing can be done, and which do not therefore call for further consideration from the clinician.

There is of course no difficulty in determining when an artery, such as the radial or temporal, is thickened: the finger trained "to feel the pulse" has no difficulty, and there need be no dubiety in the mind of the observer as to the accuracy of his observations, for it is as easy to distinguish between degrees of thickening in arteries as it is in rubber tubes. With a little practice, once attention is drawn to the matter, the smallest degrees of hypertonus are recognised by the increase in the thickness of the wall of the artery. This varying thickness has escaped observation, owing to so much attention having been given to the consideration of blood-pressure or of tension.

Thickened arteries are recognised by all physicians, and are, as is well known, of common occurrence; the real difficulty arises in determining whether the wall be thick from hypertonic contraction, true sclerosis, or perhaps even from atheroma. Atheroma is of relatively rare occurrence in the radial and temporal arteries; its occurrence is confined to aged people, and is usually associated with areas of calcareous infiltration which are easily distinguished. Sclerosis is, on the other hand, common after middle life. Before middle life it is fair to assume that uniform thickening is mainly hypertonic, unless there be discoverable one or other of the two great causes of early sclerosis, namely, chronic kidney disease or syphilis, to which I think may be added a third, namely, the use of malt and other liquors. The size of the vessel aids in the differentiation: an ordinary sized or a somewhat large vessel with a thick wall is usually structurally thickened; a small vessel with a relatively thick wall is usually only hypertonic.

Fig. 15 was taken from a section of an artery which, until a few days before death, was soft and unthickened. Some days before death it became definitely tightened up, and the figure closely corresponds with the impression made on the finger before death took place.

HYPERTONUS IN SCLEROSED VESSELS.

The description of changes in sclerosed vessels, given in the preceding chapter, has prepared the way for the acceptance of a fact of great practical moment, namely, that sclerosed vessels retain their power of contractility. The significance of the retention of this movement has been so under-valued that it is hardly referred to in the extensive literature on the circulation. It is therefore all the more necessary to emphasise the fact that arteries may be the seat of permanent structural thickening, while they are at the same time hypertonically contracted. As has already been stated in a preceding section, the idea of sclerosed and atheromatous arteries has hitherto carried with it the idea of rigidity. This is an unfortunate association, for it has led to the idea that thickened vessels are necessarily permanently thickened, that they behave in the body much as rigid tubes would behave, and that no remedial measures are available for removing such anatomical change. It is true that the structural changes in sclerosis are beyond therapeutic influence; but the hypertonus which frequently accompanies it is well within the reach of such influences. When these contentions are accepted, it will be found that the presence of various symptoms lead to the diagnosis of the hypertonic factor in sclerosed vessels and to its appropriate treatment.

Sclerosed vessels not only retain some measure of contractility, but it has seemed to me that they are abnormally responsive to some at least of the influences which determine arterial contraction. This seems to be the case particularly in old people. There is evidently a possible fallacy here, for in old people the eliminative processes are so impaired that the apparent increase of sensitiveness may be regarded, perhaps more properly, from this standpoint; the quick muscular response only indicating a more ready saturation of the blood with such waste substances as act on the vessels, the response really remaining normal. For practical purposes, it is however of great importance to know that there is in most aged people this quick and ready response, and, when a measure of confidence is acquired in recognising hypertonic contraction, any dubiety that

may exist as to the probability of such a sensitiveness will be lost.

HYPERTONUS AND RELAXATION IN ATHEROMATOUS ARTERIES.

As has been indicated in an earlier chapter the common conception of a sclerosed artery is that it is a rigid tube. This conception, as we have shown, was borrowed from the teaching regarding the vessels in advanced atheroma, and it is in accordance with experience that the extreme example becomes the standard type. This common conception is, however, erroneous, and seriously misleading. It is only in atheroma with much calcareous infiltration that the artery becomes practically a rigid tube. In my study of the arterial wall I have frequently been surprised to find the amount of relaxation that took place in a vessel that was clearly the seat of much calcareous infiltration. In other cases where, from the hooped and segmented character of the thickenings, there seemed little doubt that the thickenings were atheromatous, they could no longer be felt when the vessel became relaxed. In this latter class of case the character of the artery to the sense of touch is probably due to irregular thickenings of the intima plus hypertonic contraction, the thickenings not being felt when the wall becomes soft, by the passing off of the hypertonus, or it may be due merely to irregularity in the muscular coat itself. In other cases the rigid segments persist, no matter what measures be taken for the removal of the hypertonic contraction; and yet in a considerable number of such cases it is quite plain to the finger that a measure of relaxation can be effected. That relaxation under such conditions occurs is due to the anatomical fact, that in atheroma it is rare for the entire circumference of the vessel to be affected; there is usually a considerable part of it where there is only moderate thickening of the tunica intima; and where the muscular coat can relax and contract so as to appreciably affect the condition of the wall. The idea of the fixed and *rigid* tube has to be given up, save in extreme cases. That the recognition of such arterial changes is not due to a personal delicacy of

touch is proved by the fact that my hospital assistants and others, with whom I am brought into close association, readily acquire the skill, once their attention has been directed to individual cases and the significance of the phenomena explained to them.

The condition of the arterial wall in advanced atheroma with calcareous infiltration has been shown in Figs. 5, 6, and 7. Fig. 7 was taken from a patient with very stiff vessels, and yet I had no doubt that these vessels tightened up a little and relaxed a little under treatment.

I may here refer to two cases illustrating this point, which made a special impression upon me, from certain associations.

The first case, Mrs. M., aged 49, a patient who, simultaneously with attacks of angina pectoris, developed motor paresis, had a radial artery which felt as if composed of hard segments, which were regarded as atheromatous, and yet this character entirely disappeared, the wall becoming uniformly soft, under treatment which led to relaxation of the arterial wall. Whenever the vessel tightened up, it showed the same irregular thickening. This case is given in greater detail at p. 176.

Case 1, an Irishman, aged 56, was admitted to Ward 3, for alcoholism. On admission, he was so restless and tremulous, that it was impossible to get an absolute record of his arterial pressure; it seemed to be considerably above 200. He soon quieted down, and he acknowledged that he had been drinking too much all his life, while for the last fifteen months his drinking was only limited by monetary barriers. He drank whisky and rum, and was amused at the suggestion that he had a preference for either, seeing he was so appreciative of each. His right radial artery was thick and hard and marked by rigid segments, such a vessel as my clinical assistant spoke of as feeling so brittle that it gave one the idea that it would crack if it were fingered roughly. The pressure was 190. Under erythrol first, and then under iodide of potassium and squill, the vessel became larger and softer, and although the thickened segments were still perceptible they were not nearly so rigid, and the vessel lost that character which gave to it at first its brittle feel. Along with this change in the character of the vessel, the pressure

fell to 170. No one who followed the changes in this man's radial artery would have had any difficulty in being convinced of the accuracy of these statements. The effect on the vessel was produced by physical rest in bed and abstinence from alcohol, but mainly by the action of the drugs administered.

CHAPTER V

THE CAUSES OF HYPERTONIC CONTRACTION

1. INFLUENCE OF THE NERVOUS SYSTEM.
2. INFLUENCE OF THE COMPOSITION OR CONDITION OF THE BLOOD.
3. THE LOCALISATION OF THE ACTION ON THE VESSEL WALL.
4. INFLUENCE OF TOBACCO.

HAVING dealt with hypertonic contraction as it occurs in normal, in sclerosed, and in atheromatous arteries, we next proceed to consider the causes which determine this movement on the part of the arteries. Let me again repeat that this property separates the arteries altogether from the vulgar mechanical conception which would make them mere elastic tubes.

With regard to hypertonic contraction, the general proposition may be submitted, that it is caused by one or other of two factors:

First—The influence of the nervous system.

Second—The composition or condition of the blood.

The converse of hypertonus is either a relaxation of hypertonus or a diminution in normal or average tone.

We may now consider, in some detail, the positive aspect of this subject, which is the more important, while not forgetting that the negative has an importance of its own.

THE INFLUENCE OF THE NERVOUS SYSTEM.

Taking up first the influence of the nervous system, it is universally known that the relations of the blood-vascular

system to the nervous system have been traced and determined by long and careful experimental investigation.

The investigation of these relations has been so detailed, and the results have been so striking, that teachers, and through them students and practitioners, have almost come to regard the vessels as the toy of the nervous system,—that their manifestations merely mirror nervous influences. The effect of various emotions upon them was too apparent and too assertive to escape observation, or not to demand recognition. The path by which emotion travelled was traced, and the controlling power of the vasomotor centre in the medulla was determined. The result has been that the vasomotor mechanism thus revealed has appealed so strongly to the medical imagination that the play or movement of the arteries has been thought of through the nervous system only; as if they had no identity, as if they were the mere weathercocks of every gust of nervous influence, the bond-slave of the higher system. The nervous side of the vasomotor mechanism has been regarded as supreme; as instigating, regulating, controlling, and determining all vascular phenomena, even those attendant upon the activities and exhaustions of organs and tissues. The names of Claude Bernard, Brown-Séquard, Waller, and Schiff are "household names" in this connection. I need not dwell at length on the achievements of physiological investigation in this department; they are well known, and I have indicated that they have not hitherto been underrated. Their very brilliance has, I venture to think, seriously blinded us to the fact that physiological investigation has also shown that there is another factor which influences and determines the movement or play of the vessel wall; that factor being the composition of the circulating fluid itself. It is with this factor that I mainly deal in these pages, and I hope to show that the failure to appreciate aright this aspect of the circulation has prevented our understanding many things; while its recognition makes many things clear. I hope to show that the prevalent view is too exclusive, is incomplete, and correspondingly inaccurate and misleading.

THE INFLUENCE OF THE COMPOSITION OF THE BLOOD

It has been long known that a number of substances used therapeutically act directly upon the vessel wall,—that is, without the medium of the nervous mechanism, leading to contraction or relaxation of its muscular coat. Blake claims to have been the first to show that by injecting infusion of digitalis into the arteries contraction resulted. This observation was made as long ago as 1839. Ringer and Sainsbury's investigations are more recent, are widely known, and may indeed be regarded as having determined the views regarding the action of digitalis which we all hold. Their experimental investigations confirmed Blake's observation; but they further showed that digitalis acted independently of the nervous mechanism, acting directly upon the vessel wall. This was shown by isolating the vessels from their nerve supply.

Donaldson and Stevens, experimenting with digitalin, found that it caused constriction of the arterioles, probably through its action on the muscular coat. They further showed that it acted on the capillaries as well as the arterioles.

Haynes, investigating the action of the digitalis group upon the heart, noted that although the coronary arteries are not innervated (*sic*), squill and digitalis cause some constriction of them, probably by irritant action.

Dale, investigating the mode of action of ergot, has shown that the primary or stimulant action of this substance is a vaso-constrictor action, which is quite independent of the vasomotor centre. This investigation I shall have again to refer to.

Experiment has further shown that if blood be mixed with amyl-nitrite, chloral hydrate, morphine, quinine, or atropine, and made to pass through the vessels of a recently excised organ, dilatation of its vessels takes place: digitalin and veratrin used in the same way cause contraction.

Professor Halliburton, experimenting with choline, produced by its means a temporary fall in arterial pressure, due in part to its action on the heart, but mainly to dilatation of peripheral vessels, especially in the intestinal area. The action is due, he shows, to the direct action of this

substance on the neuro-muscular apparatus of the vessels, the same effect being produced after the influence of the central nervous system was removed. Neurine, on the other hand, causes peripheral constriction.

From these alone it would seem as if Professor Leonard Hill were over-cautious in saying that "it is conceivable that the quality as well as the tension of the blood may be the exciting cause of vascular tone." It is quite clear that there is sufficient evidence to prove that the arterial and capillary walls can be stimulated to contract by the presence of substances in the blood acting directly upon them. Therefore the composition of the blood is more than "conceivably" a factor in maintaining, influencing, increasing or diminishing vessel tone.

If we turn from the experimental side and inquire regarding the views held on the clinical side, it will be found that there is a great mass of belief that substances present in the blood affect blood-pressure.

Dr. Broadbent, later known as Sir William Broadbent, in his masterly little book on *The Pulse* gives capillary resistance as a cause of high tension, holds that certain substances in small quantities cause resistance, and mentions digitalis, ergot, carbonic acid, nitrogenous waste, and the products of imperfect metabolism as examples of such action. Broadbent, of course, knew that some at least of the substances enumerated caused contraction of arterioles and capillaries, and yet his words suggest to me that he thought of modifications in the composition of the blood as causing difficulty in its passage through the capillaries rather than of the difficulty being caused by capillary or arteriole contraction. The conception of altered composition, of something added to the blood, rendering its flow more difficult through the peripheral vessels, is practically the same view as is expressed in the term "increased viscosity," the view towards which Professor Clifford Allbutt definitely leans, while his philosophic mind and wide practical experience do not allow him to wholly adopt it.

However attractive the idea of increased viscosity may be, it is necessary to realise that there is no convincing evidence that deleterious substances, such as clinically may

be assumed to be present in the blood, so affect its viscosity or specific gravity as to impede its flow through the arterioles and capillaries. In cholera certainly, and probably in other conditions with great intestinal flux, the blood becomes so inspissated as to seriously impede its flow; but in such a condition as chronic interstitial nephritis, that there should be anything approaching this state is a very different matter. With a free supply of fluid it is difficult to think of the viscosity of the blood, thereby meaning its specific gravity, being seriously modified, seeing that fluid is so readily taken up and discharged from it.

Huchard holds that excess, and above all errors, in alimentation, throw toxic substances into the blood which produce a state of spasm of the arterial system, followed by hyper-tension and arterio-sclerosis.

Senator and many others hold corresponding views with regard to the importance of the presence of nitrogenous waste products in the blood.

The general conception undoubtedly is that "nitrogenous waste, and the products of imperfect metabolism," to repeat Broadbent's words, when present in the blood, raise blood-pressure. It is not necessary to elaborate this point further; the literature of kidney disease, and more recently of blood-pressure, is full of it, not to go further afield for examples. The explanation of the raised blood-pressure has usually been referred to increased peripheral resistance, and there the matter has as a rule been left; although, as has been said, critical and accomplished physicians like Broadbent and Allbutt have seen that this term in the equation wants determining, while to others the phrase in itself has been all-satisfying.

My contention is that certain substances present in the blood, even in small quantity, cause arterial and capillary contraction. This is the fundamental fact, and will be found to be the first step in all raising of blood-pressure which goes on to arterio-sclerosis. Even a slight degree of such contraction means peripheral resistance. There is indeed no need to go beyond vascular contraction for the explanation of peripheral resistance; it is the simplest explanation, as it is the most certain factor, and,

be it remembered, not necessarily brought about through the nervous system.

As the capillaries contract as well as the arteries, the "stop-cock" theory of Dr. George Johnstone must yield to the more prosaic view that arteriole contraction is but part of a wider contraction, and is not a special arrangement for saving the capillaries. If we are to retain any part of this attractive conception, it will require to be so modified that arteriole and capillary contraction may be regarded as a provision for diminishing the supply of an impure blood to the tissues. This is really a truer conception, for it is the good of the tissues which is the aim of the circulation, and this vessel constriction not only protects directly, but also indirectly, by producing symptoms which can lead to what we may call blood purification.

The mechanical views of the circulation have had their day, and have effected their purpose almost too well; it is now time that we should think of the vessels as *living* tubes, contracting not only under the influence of nerve centres, but under the direct stimulus of substances present in the circulating blood—contraction so caused being the *hypertonus* and the *hypertonic contraction* of our argument as much as when it is a response to nerve impulses.

THE LOCALISATION OF THE ACTION ON THE VESSEL WALL

The fact that arterial contraction can be determined by blood composition, or by blood-content, has, as has been already said, but little influenced practical medicine. It is therefore all the more necessary not only to show, as I have just done, that the proposition is supported by the weight of physiological investigation, as surely as the action of the sympathetic nervous system is established, but to see how much further physiological investigation and experiment will allow us to go.

It will be seen that experimental investigations still further warrant and support our clinical contentions; and they ought, I think, to satisfy and convince those who have hitherto thought of vessel contraction and of blood-

pressure only or mainly from the side of the vasomotor nervous mechanism.

The point which I think it is so desirable to consider is what Elliott speaks of as "the localisation of the action" of the substances which have been experimented with; that is, the particular element or part of the vessel wall which, when stimulated, leads to contraction, although the vasomotor nerve connections are severed.

The point has been investigated by means of adrenalin, the action of which as a vaso-constrictor is well known. The work of Oliver and Schäfer, Langley and others in this country, of Cybulski, Boruttau and others on the continent, have fully established the possession of this remarkable action on the part of this substance as obtained from the suprarenal glands.

Lewandowsky, investigating the action of adrenalin, suggested that its action on plain (unstriped) muscle simulated that which follows on electrical stimulation of the sympathetic nerves supplying the special part being examined. Langley followed this up, and showed that the extent of contraction of the blood vessels in the various organs varied with their control by vasomotor nerves.

Elliott has still further sustained and elaborated the preceding investigations. The conclusion at which he arrives is as follows: "The reaction to adrenalin of any plain muscle in the body is of a similar character to that following excitation of the sympathetic (thoracico-lumbar visceral, or autonomic) nerves supplying that muscle, and the extent of the reaction varies directly with the frequency of normal physiological impulses received by the muscles in life through the sympathetic nerves." As regards the action on the blood vessels, he says that "the parallel action of the sympathetic nerves and of adrenalin is maintained in the heart and blood vessels." This constriction is produced in the largest arteries as well as in the arterioles. It is not evident in the veins. From Exner and Melzer's experiments by intraperitoneal injection it seems more than probable that the capillaries, at least in certain regions of the body, are also constricted by adrenalin.

The fact of constriction being thus established, the question necessarily arises, does the adrenalin act through

the vasomotor nervous mechanism or upon the vessel wall? These, as in the case of the heart, may be regarded respectively as the neurogenic and the myogenic views. That the stimulation is produced at the periphery was proved by Brodie and Dixon, who found that the vaso-constrictor nerves to the limbs lost their electrical irritability within three hours of death, while the vessels reacted to adrenalin six hours after death. Degeneration of the sympathetic nerves after section does not hinder the action of adrenalin upon the vessels. These investigators located the point of stimulation in the "connecting link between nerve fibre and muscle fibre, . . . which is not necessarily a constituent part of the muscle fibre, nor yet of the nerve fibre," and they designate it "neuro-muscular junctional tissue."

Elliott's comment on this is that "when plain muscle develops connection with sympathetic nerves, it must at the *myoneural junction* acquire a mechanism that can receive the nervous impulse"; further, "adrenalin excites not the muscle fibre directly, but a substance developed out of it." He repeats and amplifies this as follows: "The irritability of the muscle toward adrenalin depends on the differentiation of part of its substance to form the myoneural junction. And it has been shown that the sensitiveness of reaction depends on the frequency with which it receives sympathetic nervous impulses in the reflexes of daily life. Once, however, the sensitiveness has been developed, it does not in the life of the individual become dulled by the total abeyance of arriving impulses, such as, for instance, is caused by degenerative section of the nerves. Then, indeed, the junction acquires an exaggerated irritability."

This, however, does not exhaust the interest of this subject. Another experimenter, Mr. H. H. Dale, in a most important study of the action of ergot, has shown that its primary action upon the circulatory system is stimulant,—that is to say, that it leads to contraction of the vessel wall, this being the well-known vaso-constrictor action which has led to its use as a therapeutic agent in practice. This vaso-constriction leads, of course, to a rise of blood-pressure in the aorta, and is held by this observer to be quite independent of the vasomotor centre.

Although the vaso-constrictor action of ergot is as widely

known as any fact in medicine, it is certainly not equally known that there is a further action, brought about by larger doses, which is its secondary or paralytic action. That is to say, large doses of ergot lead to dilatation of vessels instead of to constriction. When ergot has been used in sufficient quantity to produce this effect upon the vessel the vessel no longer responds to the application of adrenalin; that is, adrenalin can no longer constrict it. The action of the ergot is to paralyse the myoneural junctions on which, as has been already shown, adrenalin acts. With the paralysis of the junctions the constrictor effect of stimulating the sympathetic is also lost.

There is another gland in the body, which, as Oliver and Schäfer have shown, produces a substance which has a like vaso-constrictor action to adrenalin—the gland being the Pituitary Body, or rather its infundibular portion. The constrictor or pressor principle in this gland produces its stimulant effect directly on the arterial wall, not on the vasomotor nervous mechanism. Yet the remarkable fact has been disclosed by Dale, that ergot in large quantity does *not* neutralise its action, as it neutralises the action of adrenalin. Dale says that the constrictor or "pressor principle contained in pituitary extracts produces its stimulant effect on the arterial muscle, not through any part of the sympathetic nervous apparatus, not through the related structures on which adrenalin acts, but through some other substance or substances of the muscle fibres themselves."

It is therefore plain and evident that vessels constrict by the direct influence of substances in the blood. The conception which has so long dominated our views, and it is hardly too much to say sealed our eyes, that vessel contraction or relaxation, or the associated alterations in blood-pressure, are the manifestations of nervous impulses, has to be given up in great part, and must share its honours with the other factor.

While the direct influence of constrictor substances on the vessel wall is thus established, it is to be noted how subtle the processes are: adrenalin acts upon what is neither nerve nor muscle, but is euphoniously termed the *myoneural junction*; pituitary extract acts on the vessel wall as adrenalin does, not, however, upon nerve, nor upon the myoneural junction, but upon some substance which, as it is none of the things mentioned,

is assumed to be muscular. Choline acts in the same way, but its action can be prevented or neutralised by atropine.

How futile seem the controversies regarding the neurogenic or myogenic origin of vascular response! How many controversies have been waged over alternatives, when each was but part of a more central truth!

THE INFLUENCE OF TOBACCO

In Chapter XV., which deals with angina pectoris, it is pointed out that one of the recognised clinical varieties of the affection is the toxic form. I illustrate this form by reference to the effect an overdose of tobacco may have on myself. The cardiac discomfort accompanying the hypertonic contraction noticed in the radial artery illustrates what is perhaps the mildest form of angina pectoris that can be experienced. It is, however, difficult to determine through which system tobacco acts, so I give it this special paragraph here. Tobacco is much used, and its influence upon the vessels has definitely attracted the attention of clinicians. Personally I have not so far paid any special attention to it, but my clinical assistant, Dr. J. L. Green, has recently made observations on himself and a friend. He tells me that if he or his friend inhales a cigarette made of Virginian tobacco, the hypertonic contraction of the radial artery which ensues is very marked, and that with the hardening of the vessel the hæmomanometer reading rises 15 mm. Hg. Here, as in other instances, the *fact* is the constriction of vessels. What the blood-pressure may be inside the constricted vessels is a *problem* dealt with in other chapters.

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CHAPTER VI

THE CLINICAL ESTIMATION OF BLOOD-PRESSURE

1. ARTERIAL TENSION AND BLOOD-PRESSURE.
2. METHODS OF ESTIMATING BLOOD-PRESSURE.
3. THE FACTORS WHICH DETERMINE HÆMOMANOMETER READINGS.

ARTERIAL TENSION AND BLOOD-PRESSURE.

FOR many years the attention of clinicians has been largely directed to what has been called "arterial tension." The significance to be attached to the term has been variously interpreted; its relation to blood-pressure has been discussed by Professor Clifford Allbutt and others. In its everyday and ordinary use there is no doubt it has been associated with much confusion of idea and serious error. Allbutt, with his critical faculty, decides that the word "tension" is practically not applicable to the blood inside the vessel, and with this I entirely agree. If it is not to be referred to the blood, it might be assumed that it is applicable to the vessel wall. It is not, however, proposed to attempt to discuss the various shades of meaning the word "tension" may have,—that may be left to the pundits in physics. And yet I cannot leave the matter without expressing my belief that the term was used by our most expert clinicians to express not blood-pressure, nor thickness of vessel wall, but the relationship between blood and wall —the sustained fulness of the vessel, and so forth.

More recently, attempts have been made to do away with the term "arterial tension." The present writer was amongst the first to definitely propose this, but it is almost impossible to get a familiar word given up. Others have met

the difficulty by maintaining that the terms "tension" and "pressure," are synonymous. Personally I hold that whatever conduces to clearness of thinking is desirable, so I am quite ready to accept this definition of the terms, but I do not think that it is correct historically. It seems to me that our best clinicians meant more than this indicates, that to them there was a living and active relationship between the state of the vessel wall and the volume of blood inside it. Dr. Leonard Williams, in a lecture delivered at the Medical Graduates College, says: "High arterial tension is an expression which is used as a synonym for 'high blood-pressure,' than which it is more euphonious, but less correct." While believing, as I do, that our ablest clinicians meant more than is here implied, the meaning of words is frequently modified, and it would greatly simplify the position were the term "tension" to be given up, or definitely used as synonymous with "blood-pressure."

In the examination of the pulse two factors are commonly considered, no matter what terms the observer uses to express his mental concept, namely, the condition of the arterial wall and the blood-pressure inside it. Putting aside for the moment the first of these, the estimation of the pressure within the artery is undoubtedly taken as the index of the power of the heart. The "strength" of the pulse has no meaning clinically, save as an index of heart power. A "feeble" pulse means to the clinician a feeble heart; a "strong" pulse the reverse. That the pulse can reveal this is the warrant for "feeling the pulse," which is the custom of the clinician.

The effect of the blood-pressure upon the arterial wall could, I presume, be expressed in terms of "degrees of tension," and it was the cultured appreciation of this which, I believe, marked the skill of our ablest physicians; but it can easily be that the acquisition of this skill is now regarded as too difficult, for the internal pressure, whatever it be, is exercised upon tubes which vary in thickness of wall, in size of lumen, and in the relation of wall to lumen. The term "tension," unless only used as a somewhat rough indicator, has thus a very subtle significance.

Another aspect of this question may be referred to,

namely, the not uncommon use of the term "high tension" to thickened radial arteries. This confusion arose from the teaching that high blood-pressure and thickened arteries went together; so, when the arteries were thickened the pulse was frequently spoken of as of high tension. Of course, this error was not universal, for it was widely recognised that in thick radials there might be a feeble pulse of low blood-pressure. I refer to this because it is by recognising how error originated that it is most readily rectified.

Senator, in a recent lecture on arterio-sclerosis, says that in his experience an increase in arterial tension is a very inconstant factor, and depends greatly upon the condition of the heart; while the rigidity of the arteries places a serious obstacle in the way of determining the conditions of pressure within them.

Thickening of the arterial wall no doubt adds to the difficulty of estimating the blood-pressure; but it is, I think, not so difficult as is the estimation of wall tension. As it is blood-pressure, in the sense in which it has just been indicated, which is the main object aimed at when the pulse is examined, it materially helps if this single conception be kept before the mind, and all thought of "tension" be abandoned. That the finger can be educated to a high degree of proficiency in determining blood-pressure there can be no doubt. What has to be acquired is the power to estimate the pressure *inside* tubes of different sizes, and with walls not only of different but of varying thickness. As we learn to distinguish between large and small objects, as we distinguish between a thick-walled and a thin-walled tube of equal circumference, so we learn to judge of the flow of fluid through tubes. There is nothing more subtle or mysterious in it than this, but it probably requires a delicate finger and much careful self-education. Could we introduce a cannula into a patient's artery, and connect it with a manometer, a record would be obtained which would check observations made by the finger, and provide a record the accuracy of which would not be questioned. As this cannot be done, methods have been devised and instruments have been constructed with the object of providing such records. In all skilled work ingenuity is constantly directed to devise means of

eliminating the individual equation: in the particular matter with which we are dealing it would be of value to be able to do this. It would be of still greater value could an electrical balance be devised that would correctly weigh and estimate the data upon which a diagnosis is based, so that the individual judgment and the personal mental bias would be eliminated. We are, however, far from this, and after we have counted blood corpuscles, estimated hæmoglobin richness, determined blood specific gravity, found the total nitrogen excretion, taken sphygmographic tracings, used the most recent of "blood-pressure" instruments, and piled up a goodly array of clinical data, the final judgment may be no whit better. The personal equation cannot be eliminated in diagnosis, and when it is all-important to form a correct estimate of the circulation as a whole, our most recent methods still leave the decision to the individual acumen and experience; to what is nothing else than the skill of the individual, determined by inherent mental faculty, developed by education and enriched by much accurate observation.

METHODS OF ESTIMATING BLOOD-PRESSURE.

We may now proceed to examine the methods in use for the clinical determination of blood-pressure, to note some of the contentions based upon the records so obtained, to determine what data the methods used *really* supply us with, and what practical use can be made of the data which they supply. I have no special desire to pull down if I cannot at the same time build afresh, but it is more than time that this important subject were put upon a sound foundation, and that the errors with which it threatens to be surrounded, it might even be said adorned, should if possible be checked.

This is not mere hyperbole, for the present position is not only curious but undesirable. The position is this: on the one hand may be placed the physiologists who have accepted the instruments in use, and believe that they give an accurate record of blood-pressure; along with them there are a certain number of physicians who have accepted the dictum of the physiologists, and have drawn important clinical inferences from the rigid application of physiological methods

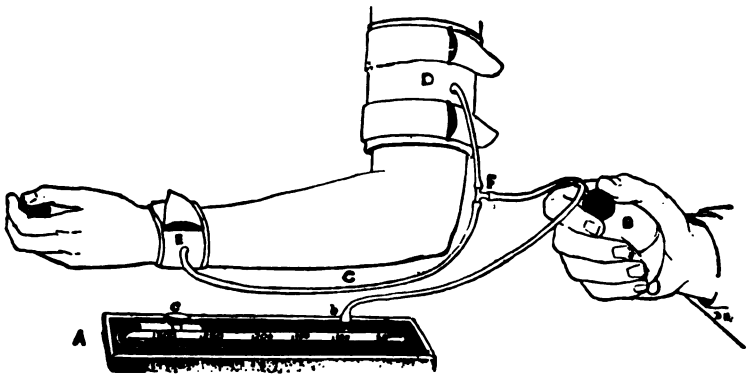
to pathological conditions. On the other hand, there are a number of physicians who have absolutely discarded the instruments in question, not only as useless, but as seriously misleading. When this is the state of opinion, it is evident that there is something to be found out, and the matter is so very important from the physician's standpoint that I do not hesitate to submit my observations and conclusions, in the belief that they can and ultimately will reconcile divided opinion.

The plan or principle on which blood-pressure instruments are based is simple, and may be briefly described. An armlet made of some strong material has a rubber bag attached to its internal surface. When this armlet is applied and fastened by means of straps, the rubber bag is blown up by means of a ball syringe attached to the bag by rubber tubing; another piece of rubber tubing leads to a manometer which records the pressure of the air inside the apparatus as it is slowly pumped in. When the bag of the armlet is distended sufficiently to grasp the arm, the index of the manometer moves up and down with every arterial pulse wave: at a certain pressure the pulse wave, as seen on the index of the manometer, attains its maximum degree of excursion; this point is taken by some observers to represent the *diastolic pressure* in the arteries. Other observers use the same kind of instrument in another way,—the bag of the armlet is distended until the arterial circulation in the arm is stopped by the pressure of the distended bag, the stoppage being judged of by the finger on the radial artery, or by a second bag attached to a wristlet. The height to which the manometer index has risen is taken as representing the maximum or systolic pressure. A third method is applied in the construction of Gärtner's tonometer and corresponding instruments. In this method one of the fingers is rendered bloodless by passing a tight rubber ring from the tip of the finger upwards along the two terminal phalanges, then a rubber bag is passed round the proximal phalanx and distended with air sufficiently to ensure sufficient compression to prevent blood passing into the finger after the rubber ring is removed; the pressure in the bag is recorded by a manometer attached to it by a rubber tube; the pressure is slowly

lowered, and the point at which the index stands when the finger flushes is taken as the systolic pressure.

In the first method the maximum pulsation obtained from the compressed vessel is taken as the record; in the second and third methods the point at which the vessel or vessels are so obliterated as to arrest the circulation in the part is taken as the record.

It is these records which it is maintained give the "blood-pressure." After using and comparing various types of instrument in a variety of cases it seems to me that those which are used to obtain systolic pressure by



Dr. George Oliver's hæmomanometer as made by Mr. Hawksley, 357 Oxford Street, London, W. A detailed description of the instrument and how to use it is supplied by the maker.

means of obliterating the brachial artery, or its branches in the upper part of the forearm, are the most satisfactory. They are mostly modifications of the Riva-Rocci instrument. Personally I prefer Dr. George Oliver's modification for two reasons: first, because in the manometer a spirit index is used instead of mercury, which makes it more easily carried about; and second, because it has a wrist bag by means of which you can *see* when the pulsation in the radial artery stops, which is less of a strain upon the attention of the observer than using the finger, while those around the patient can have the whole proceeding demonstrated to them, which is impossible when the observer can only communicate what his finger determines.

By the methods which have been described it is apparent

that when, for example, the armlet is applied to the upper arm, pressure is exerted by the air-pad upon all the soft tissues of the arm, sufficiently to compress the brachial artery, so that by obliteration of its lumen the pulsatile movement is arrested on the distal side of the compression. It is surely reasonable to claim that this can be expressed as *compressibility*, and that the soft tissues take some share in the result; but above all, it would seem reasonable to assume that the physical characters of the artery will materially influence the result. That it is compressibility which is determined cannot really be denied; and this being so, it is a matter of common knowledge that the compressibility of a tube depends upon the thickness of its wall, and the relation between that and the size of its lumen. A large tube with a thick wall is more easily compressed than a small tube with the same thickness of wall. And yet, plain as this may appear to be, it is taught that the compressibility of an artery is determined wholly by the pressure of the blood inside it, that the thickness or thinness of the wall is a negligible factor, and that therefore the compressibility, as measured by the hæmomanometer, is literally "blood-pressure."

Janeway, in his well-known book on blood-pressure, definitely states his view as follows: "That a sclerotic vessel may offer considerable resistance to compression is a common belief which I do not think is justified."

It is unnecessary to single out others by name, for it will be found in most recent writings that compressibility has been assumed to be "blood-pressure,"—no other explanation or interpretation of records has been given, so far as my reading has revealed. No doubt, as Janeway says, there is a "common belief" which is contrary to this, but the object of writers on blood-pressure seems to be either to ignore it or to assume that it is one of the *common beliefs* which possess the minds of uninformed persons.

Although the condition of the arterial wall is so definitely assumed to be a negligible factor in the records obtained by means of the hæmomanometer, it must be remembered that, before the instruments referred to came into such extensive use, it was fully recognised by the best clinicians that the state of the vessel wall was an important

factor in what was commonly known as "compressibility of the pulse." It was, indeed, universally recognised that when the wall was thickened the pulse was less compressible. If the new conception were correct, it would imply that what was formerly regarded as knowledge was a mere figment of the imagination, and that all our knowledge about thin and thick tubes appertained to the physical characters of rubber tubes, and had no relationship to the arterial tube. The revolutionary nature of the newer view seems to have been but partially realised. It seems to have been based upon observations on normal walled vessels, for I cannot imagine anyone accustomed to the clinical observation of arteries, and equally familiar with the appearance of the same vessels after death, accepting the new view. It is as unsound pathologically as it is clinically. If the wall of an artery can become many times thicker than the normal, the common physical law must be applicable to it,—it cannot but be more resistant, it cannot but require more power to obliterate its lumen. The normal radial artery, for instance, collapses by its own weight when empty, while it is commonplace knowledge in pathology that if the wall of an artery is thickened its cut end gapes. Certain physical properties are as applicable to arteries as to rubber tubes. It is indeed curious to note, on the one hand, the application of the physical characters of rubber tubing to illustrate and elucidate the phenomena of the circulation; and on the other hand, the apparent abandonment of all our common knowledge of physics when the compressibility of an artery is under consideration.

As the result of experience, I venture to say that when the method of obtaining hæmomanometer readings is submitted to the physicist, he will smile at the suggestion that the readings represent the pressure of the fluid inside the compressed vessel. This point need not be further elaborated, and I willingly turn from it to the constructive side of the subject.

THE FACTORS DETERMINING HÆMOMANOMETER READINGS

I submit that, by the methods used, the light of a limited knowledge of physics suggests that there are three

factors to be considered—(1) The soft tissues surrounding the artery; (2) the arterial wall; and (3) the pressure of the blood within it.

Tissues surrounding the Artery. — Taking up the first of these—the soft tissues surrounding the artery—I may at once state that so far as I can see this factor is practically negligible. I thought at one time that it might prove to be an important factor, but after a careful comparison of the girth of arms, whether due to muscularity or adiposity on the one hand, or to emaciation on the other hand, with manometer readings, I am convinced that the factor is, as I have said, practically negligible. Even the application of the pressor bag over the arm covered with ordinary clothing gives much the same record as when applied over the uncovered arm. Further, the bag on the upper part of the forearm gives practically a like reading to that obtained from the upper arm. I therefore pass from this factor to the consideration of the other two.

Thickness of Arterial Wall.—That the thickness of the wall of a tube, and the proportion this bears to the bore, affects its compressibility is a fact of common knowledge, as has been already pointed out. It is one of the beliefs it is difficult to realise should require proving. That the contrary opinion exists, when the tubes are arteries, has been already shown; and it seems to me that it has arisen by the rigid application to clinical and pathological problems of the conclusions drawn from physiological experiment and observation. It has come about in this way. A normal artery of such size as the radial has such a thin wall that, when empty, it collapses, as a piece of thin rubber tubing collapses; if the vessel be full of blood, or other fluid, its walls are held apart, and its lumen is maintained. If by any device fluid pressure is exerted on the outside of this full vessel a very small extra pressure will lead to its collapse. No matter what the pressures be, the result is, I believe, the same. The wall is practically, if not theoretically, negligible. That similar results have been obtained with arteries which have undergone what I have defined as arterio-sclerosis is to be explained by the entire loss of tone in the dead vessel, so that it is no longer comparable to the living vessel.

There is, of course, no doubt that the arteries we examine clinically vary enormously in the thickness of their wall, and the relation of thickness of wall to bore. There are, however, no records of the results of the examination after death of radial arteries known to be thickened during life. Pathologists and clinicians in the post-mortem room have gone to the aorta or the cerebral arteries in search of atheroma, and have left the vessel felt during life undisturbed in its resting place, and overlooked even amidst the yearnings of clinical curiosity. This was formerly my own position, but in 1901 I drew attention to the gravity of the omission; and what I wrote then and subsequently is the outcome of observations which are almost monotonous in their uniformity.

The difference in the thickness of radial arteries and in the relation of thickness of wall to bore has been shown in Figs. 9 and 11, and is further represented in Figs. 14, 15, 16, 17, 18, 29, and 30.

There is no question here of post-mortem rigidity, and the influence of fixatives; these arteries felt after death as they had felt during life, save for the absence of pulsation in them, so that the only question is what changes led to their thickening? These changes I have described in an earlier chapter. No fixative treatment of normal arteries will produce such pictures as I have given. Professor MacWilliam found that arteries taken from amputated limbs were by stimulation so altered that their diameter could be reduced to one-third, with, of course, a corresponding thickening of the wall. This is a wide range of contractility in a normal vessel, yet in arterio-sclerosis, where the thickening encroaches upon the lumen, and contractility is retained, the diminution is still greater. Some of my cases have shown a thicker wall and a smaller bore than MacWilliam records. I cannot but think that those who have thought that the vessel wall was negligible have not had the data necessary to a correct opinion. The following chapters will illustrate the part taken by the vessel wall in hæmomanometer observations. It is, however, necessary to repeat what has been said earlier, that thickness of the arterial wall may be due to two factors, separate or combined, namely, permanent structural thickening and hypertonic contraction:

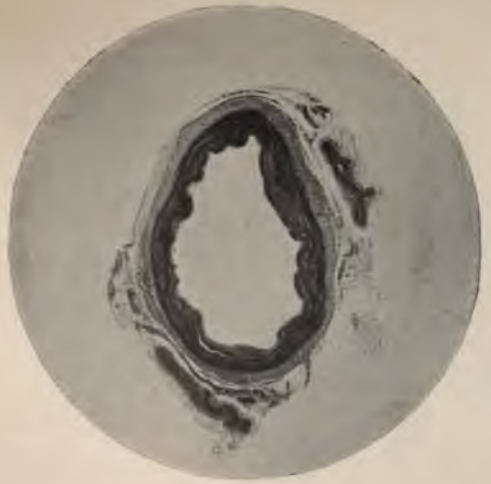


FIG. 14.—Radial artery about normal size, showing slight hypertonic thickening and some degeneration of media (×16).

FIG. 15.—Radial artery of normal size, showing some hypertonic thickening (×15).

FIG. 16.—Radial artery, showing hypertrophy of the media, slight thickening of intima—no hypertonic contraction (×15).

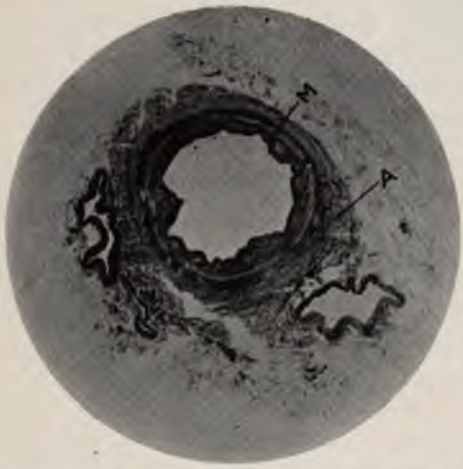


FIG. 17.—Radial artery, showing hypertonic contraction; **M**, thickened media; **A**, thickened adventitia (×15).

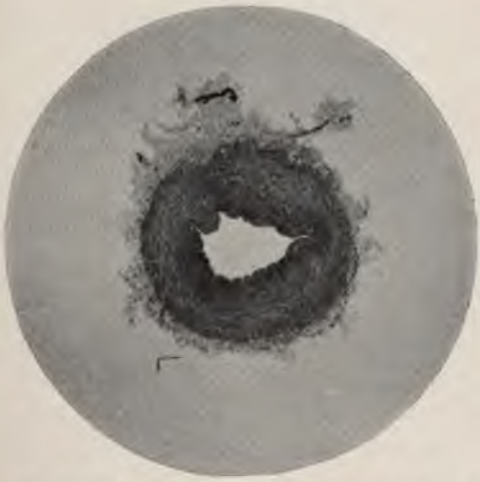


FIG. 18.—Radial artery, showing pure hypermyotrophy and hypertonic contraction.

and if anyone remains sceptical of the occurrence of hypertonic contraction let me again refer to Professor MacWilliams' observations, that in an amputated limb the arteries could by stimulation be made to contract so as to reduce their lumen by two-thirds. I merely contend that arteries do this in the living body; and if clinicians have not trained themselves to note its occurrence they will find it a most interesting study and an invaluable guide.

The Blood-pressure inside the Vessel.—It is one hundred and seventy-five years since Stephen Hales introduced the study of blood-pressure by putting a tube into the crural artery of an animal. Ninety-five and one hundred and fourteen years later, Poiseuille and Ludwig respectively connected a mercurial manometer and a writing style to the tube. Others have perfected the method thus begun; but it is unnecessary to trace here the stages of physiological evolution to its present high level of experimental exactitude. It is enough for our present purpose to know the conclusions at which physiology has arrived. No higher authority can be found than Professor Leonard Hill, and he places the pressure in the brachial artery of a healthy young man at from 110 to 130 mm. of mercury. Faivre, quoted by this authority, measured it directly in man during the amputation of a limb, and his results gave a pressure of from 110 to 120 mm. Hg. This is in close accordance with the readings obtained by the use of the hæmomanometers already referred to. My own observations in persons with perfectly soft arteries place it a little lower, namely, at from 105 to 115, and not above 120. At this point, however, I am in the happy position of being practically in agreement with physiological teaching. I can draw further from the same source, but would emphasise the facts, namely, that during sleep and when the body is kept at rest, the pressure is 20 mm. lower; and that it rises 20 mm. Hg. after violent exercise. It can therefore be stated that, in the normal state of living, the pressure ranges from 90 to 120; and after violent exertion may rise to 140, this extra rise passing off in about fifteen minutes after exertion stops. These figures closely correspond to my own observations. I have, however, made observations on one active person whose pressure varied

between 85 and 105, but this is an exceptionally low reco for health.

On the strength of the physiological statements, I subm that the 20 mm. Hg. of rise after violent exercise marks t limit of the reserve of power of the normal average hear This is the amount of rise of pressure that the heart can co with ; if raised somewhat above this the left ventricle woul give way, and acute dilatation would result, as clinically w know sometimes occurs. The rise of pressure is not primaril due to heart action, as seems to be the common conceptio The rise of pressure is due to peripheral constriction cause by muscular compression on capillaries, arterioles, and eve arteries ; the inevitable and unavoidable result being a raisin of pressure in the larger arteries and the aorta. This raisin of pressure is purely mechanical, and takes place in a syste of rubber tubes, just as it does in the blood vessels. Th living factor steps in when the heart is stimulated to mee this raised aortic and arterial pressure by increased worl shown in more rapid and stronger ventricular contraction The length of time the left ventricle can maintain thi depends on the quality of the individual heart. If the le ventricle cannot rise to the aortic pressure and open th aortic cusps there is a syncope, which may be and often fatal. This may happen presumably with a rise short of 2 mm. Hg., and here at the outset we see that clinical medicin and pathology branch off from the physiological standar The physiological doctrine that a healthy young man ca safely do what will raise his aortic pressure to 140 mm. H is accepted, but the two words " healthy " and " young " mus never be omitted from the proposition. The observation of clinical pathology, however, are not confined to the health or the young, although it seems as if some people though that a physiological truth was one of those eternal veritie such as that $2 + 2 = 4$, applicable in all circumstances and a all times. It is not safe to raise the blood-pressure in a aortas by this extra 20 mm. Hg. : and when we get hæmo manometer readings in man from 160 to 260, and eve up to 300, we turn to physiology and ask, " Is this blood pressure in the sense in which you use the term ? " I kno that in experimental work the aortic pressure can be some

thing like doubled by constricting arteries and capillaries, but to prevent the heart coming to a standstill under these conditions the vagi have to be cut.

In the consideration of blood-pressure it must be remembered that the heart and the vascular system have a self-protective nervous mechanism. If the aortic pressure is raised by systemic constriction it can be relieved by splanchnic dilatation, and conversely. If the aortic pressure be higher than the left ventricle can fully overcome the residual blood in the ventricle is increased ; this leads to over-distension ; over-distension acts upon the vagal terminals, and the heart is slowed, to give time for the hypertense aorta to be relieved by the passage onwards of more blood. The heart is also saved by blood accumulating in the veins when arteries constrict. In this way it follows that less blood reaches the left ventricle, and, as was shown by Hales one hundred and seventy-four years ago, "the real force of the blood in the arteries depends on the proportion which the quantity of blood thrown out of the left ventricle in a given time bears to the quantity which can pass through the capillary arteries into the veins at that time." The circulation has therefore very efficient ways of preventing pressure becoming too high, and yet we are told that blood-pressure rises with age, that in fact high blood-pressure is a common sign of senility ! This is the outcome of applying an instrument, evidently physiologically accurate, all along the line of pathological change, and insisting that the instrumental readings continue to have the same value. I have heard a physiologist describe what would happen if blood-pressure rose to 300 mm. Hg. I do not repeat his words, but it amazed him to be told that such readings were obtained from the hæmomanometer.

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CHAPTER VII

A NEW SCHEMA OF THE CIRCULATION TO ILLUSTRATE—

1. QUESTIONS CONCERNING BLOOD-PRESSURE.
2. THE INFLUENCE OF THE ARTERIAL WALL IN HÆMOMANOMETER READINGS.

AFTER I had arrived at what appeared to me to be reasonably definite conclusions regarding the clinical phenomena, *first*, of hypertonic contraction of arteries and its effect on the blood-pressure in the contracted vessels; and, *second*, of the influence of the arterial wall in hæmomanometer readings, it seemed to me desirable to investigate these points from the physicist's standpoint.

I was fortunate enough to arouse the interest of Dr. Cargill Knott, Lecturer on Applied Mathematics in the University of Edinburgh, in these problems, with the result that a schema of the circulation was devised which is represented in the accompanying figure. The schema was the outcome of much patience extended to me, and of Dr. Knott's technical skill in his own department. In its completed form it seemed to meet both his and my requirements.

The two points to be investigated were—*First*, what effect was produced by constriction of arteries upon the pressure of fluid inside them? *Second*, what part did the arterial wall take in determining arterial compressibility, and therefore in determining hæmomanometer readings. These two points seemed to me to belong almost to pure physics, although there were factors in the living circulation which they did not embrace.

The schema consists of a glass reservoir R^1 with three glass arms to which large rubber tubes are attached; these

are succeeded by rubber tubes about the size of the brachial artery: these are again succeeded by larger tubes which are attached to the three glass arms of the second glass reservoir

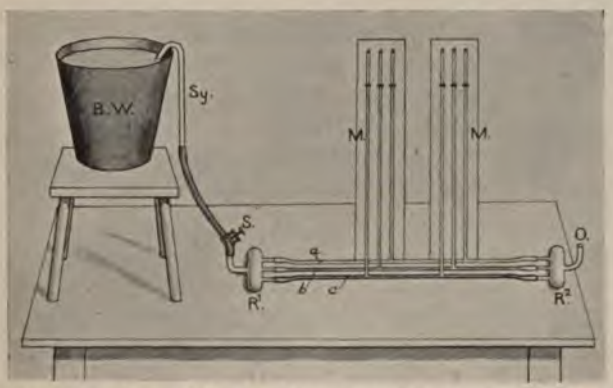

FIG. 19.—Schema of the circulation devised by Dr. Cargill Knott and the author; description in text. The thick tubes between tubes *a*, *b*, *c* and R^1 and R^2 are omitted from the figure.

R^2. On each of the three smaller tubes two glass gauges M are introduced to indicate the pressure inside the tubes. The

FIG. 20.—Photographs of transverse sections of the three tubes used in the scheme.

three tubes were made *nominally* of equal bore, but of different thickness of wall. This ideal was not realised; but, as they are, they suffice for our purpose. Sections of the tubes are shown in Fig. 20,—A having the thinnest wall, B a medium thickness of wall, and C the thickest wall with the smallest bore or lumen. It will be seen, by comparing these with the sections of arteries, that there is no greater difference between the tubes than between the arteries.

Water is made to flow through this schema either by means of a syphon Sy. connected with a bucket of water

B.W., or the thick rubber tube leading to reservoir R^1 can be attached to a water tap. The pressure can be regulated as desired, by raising or lowering the bucket, or by means of the screw S. The pressure can be regulated by means of the tap when the schema is attached to a water pipe. When water is flowing through this system the two reservoirs R^1 and R^2 ensure a uniform pressure in the three tubes *a*, *b*, *c*; at all events, the reservoirs, in the view of the pure physicist, remove possible sources of fallacy; and this is desirable, as the problems which surround the flow of fluid through tubes are very intricate.

When water is flowing through this system, say from a bucket of water, there is a steady fall in pressure from the syphon Sy. to the outlet O. This fall is shown by the gauges M on their respective tubes *a*, *b*, *c*. The *mean pressure* in such a system is the average between the highest and the lowest pressure, and it is practically the same in all three tubes.

If now any one of the tubes be constricted, no matter how slightly, the pressure immediately falls on the distal side of the constriction, and rises on the proximal side. The *mean* pressure in the whole system remains unaltered, but the *distribution* of the pressure is changed.

The scheme is so sensitive that touching any of the tubes at any point alters the level of all the six gauges; the water rising in *all*, save the one or two on the distal side of where the finger is applied—in them the water falls.

It follows that in and beyond the constricted area pressure falls; above it, it rises. This is a physical law, and must be as applicable to arteries as to rubber tubes. There appears to be no warrant for the view that there is a rise of blood-pressure inside constricted vessels.

The *second point* to investigate was the difference in the amount of pressure required to obliterate the lumen of the tubes *a*, *b*, *c*, with their different thickness of wall but with the same internal pressure. This was first roughly tested by means of weights applied over a cork resting on the tubes in succession. The cork was attached to an upright rod kept in position by means of two wire loops attached to a suitable block of wood. Roughly, tube A required 400 to 500 grains

to obliterate its lumen; tube B required about 1500 grains; and tube C required about 4000 grains. The conditions affecting the pressure of the fluid was uniform in all, and yet there were these enormous differences in the amount of pressure required to obliterate the lumen of the three tubes. The diameter of the lumen of the tubes and the thickness of their walls is shown in Fig. 20.

It was, however, desirable to be somewhat more precise in our observations, and I continued observations on a new plan. When the pressure in the gauges was at zero, that is to say, when the system was full, and the water at the foot of the gauges, I attached a manometer to the top of one or two of the gauges and closed the tops of the others. I then turned on the water, and found that I could make investigations with any internal pressure I liked, so long as it was not so high that it burst the tubes or forcibly separated the attachments. I found that I could work conveniently with a pressure as high as 30 mm. Hg. It then occurred to me that I had found a use for the hæmodynamometer of Oliver, which, as an instrument of clinical use, I had discarded for his hæmomanometer. By means of this instrument I could with sufficient accuracy determine in mm. Hg. the amount of pressure required to obliterate the respective tubes.

Before giving the figures, I would point out that tube A was so thin walled that it collapsed with its own weight, as a normal artery collapses, while the other two tubes always retained their round form.

The internal pressure, as shown by manometers attached to the top of some of the gauges M while the others were closed, was 30 mm. Hg. The pressure required to obliterate the lumen of the tubes as measured by the hæmodynamometer was as follows:—

A tube	B tube	C tube
10 mm. Hg.	40 mm. Hg.	84 mm. Hg.

These numbers are at least approximately correct, and show roughly the same proportion to one another as the rougher method showed.

It is interesting to note that in A, where the tube was only kept open by the contained fluid, the lumen of the tube

was obliterated by a directly applied pressure of 10 mm. Hg., while the pressure of the flowing fluid inside it was 30 mm. Hg. It followed from this that the pressure of the flowing water in the other tubes only required to be allowed a like measure; so that in B the tube wall equalled 30 mm. Hg., while in C it equalled 74 mm. Hg.

That this represented, at least approximately, the part taken by the wall of the tube, was confirmed by the further observation that, when the internal pressure was raised 10 mm. Hg. higher, the pressure required to obliterate the lumen was also raised 10 mm. Hg. This observation was not extended to tube A, as a sustained internal pressure of 40 mm. Hg. found out all the weak points in that tube.

It appeared from these results that the general opinion was correct, that the compressibility of a tube was determined partly by the pressure of the flowing fluid inside it, partly by the thickness of its wall, and the relation between that and the lumen of the tube.

Objection has been taken to these results on the grounds that arteries are not rubber tubes.

I have made some observations with vessels obtained from the post-mortem room, and, so far as I have yet carried these, they seem quite worthless, for the simple reason that the dead vessel no longer possesses the *tone* or the elasticity of the living vessel. In fact, the rubber tube is a better substitute for the living artery than the dead one is. It may be that when I have the opportunity of testing some of the thickened brachial arteries which I have felt during life, and which always give a high haemomanometer reading, I may modify this view; but from what I have seen, I am not sanguine that the results obtained after death will in this particular help towards the elucidation of the clinical phenomena.

A *third observation* is worthy of being recorded, although it had no direct bearing upon the two points which were specially investigated. The observation was as follows:— Working with an internal pressure of 30 mm. Hg., when the lumen of tube A was obliterated, the manometer on the proximal side of the point of constriction showed a rise from 30 to 100 mm. Hg.; the tube became much distended, and

gave way at every weak point. Tube B when its lumen was obliterated showed a rise from 30 to 95 mm. Hg. Tube C showed a rise from 30 to 42½ mm. Hg. when similarly treated. These may be tabulated as follows, — with an internal pressure of 30 mm. Hg., when the tube was obliterated there was a rise of in

A	B	C
70 mm. Hg.	65 mm. Hg.	12½ mm. Hg.

This observation has considerable practical significance. In the first place, it shows the great rise in pressure that would take place in arteries the size of the radial if they did not contract along with the arterioles and capillaries. The relatively small rise that took place in the thick-walled tube C was very striking.

The phenomena shown by means of this scheme enable us also to understand very clearly the circulatory changes which occur in the brain. The scheme can further be utilised for teaching purposes to help students to understand and to appreciate the questions of internal pressure and of thickness of arterial wall.

CHAPTER VIII

PRESCLEROSIS - HYPERTONUS *VERSUS* BLOOD-PRESSURE—THE MODE OF PRODUCTION OF ARTERIO-SCLEROSIS

PRESCLEROSIS-HYPERTONUS *VERSUS* BLOOD-PRESSURE.

THE term *Presclerosis* has been introduced by Huchard to indicate the vascular condition which precedes arterio-sclerosis. I have already referred to Huchard's views regarding the meaning of the term arterio-sclerosis, and shown that he does not separate the condition from atheroma, as I contend must be done if a clear and unambiguous conception of the course of arterio-sclerosis is to be formed. It is therefore difficult to be sure that one is forming a precisely accurate estimate of his opinion on certain points. He uses, for instance, the terms *arterial hypertension* and *blood-overtension* as synonymous. I have in an earlier chapter dealt with the confusion which surrounds the use of the former of these terms, and it does not seem to me that Huchard has simplified the problem. He quotes with approval Mahomed's study of the pre-albuminuric stage of Bright's disease, in which he recognised a functional period characterised by blood hypertension. Rosenbach also is in accord with the view that the thickening of the arterial wall is the result of the pressure of the blood. The fundamental idea is increased blood-pressure, at one time called blood-hypertension, at another arterial hypertension; while Dr. Leonard Williams, who is appreciatively referred to by Huchard, and has been referred to in an earlier chapter, unambiguously asserts that the terms are synonymous. I have already expressed a doubt as to the accuracy of this, for it always appears to me that the writings of Broadbent,

Clifford Allbutt, and Huchard himself show that they distinguished between the blood-pressure and the vessel wall in a way that Leonard Williams does not, and that this explains Broadbent's belief in the finger for the examination of the pulse, while Williams regards it as a most inept and fallacious medium as compared with a hæmomanometer. In fact, in the writings on this subject there is often apparent either a break in the harmony or a false note, which is unavoidable when a factor is present which is at one point acknowledged in the abstract but ignored at a further point in its exposition.

To Huchard the stage of *presclerosis* is the period during which there is a continuously maintained elevation of blood-pressure. So much may be accepted as correctly interpreting his position, but he follows others in attributing somewhat vaguely the raised pressure to vessel constriction. I cannot better illustrate Huchard's position than by referring to the discussion of a recent paper which he submitted to the Académie de Médicine of Paris. In this discussion Lancereaux and Chantemesse both denied that arterio-sclerosis was the result of hypertension; maintaining, on the other hand, that it was of the same nature as the anatomical changes met with in gout, rheumatism, and lead-poisoning, that indeed "it was manifestly of toxic origin," as the latter of these expressed it. No more complete justification could be found of my contention that the whole subject of arterio-sclerosis, its clinical significance, and the current methods of estimating blood-pressure required clearing up.

In the discussion referred to it is apparent that Lancereaux and Chantemesse confine their conception of arterio-sclerosis to changes in the tunica intima; while Huchard's conception of the term, already dealt with in Chapter II. of this book, is equally incomplete. The combination of changes to which I hold the term ought to be confined is nowhere explicitly recognised, and could hardly be, seeing that the fundamental and primary phenomenon of hypertonic contraction has been overshadowed by the idea of raised blood-pressure.

Senator thinks that the early beginnings of arterio-sclerosis cannot be recognised clinically with certainty.

THE MODE OF PRODUCTION OF ARTERIO-SCLEROSIS.

The whole problem seems to me to be at once clarified and simplified when we begin with the first clinical step in the process, namely, hypertonic contraction of the arteries with which we clinically deal. It is not that this arterial contraction has been altogether ignored, but its pre-eminent importance has not been realised. Huchard recognises that toxic substances in the blood possess convulsive properties which act on the vascular musculature, producing in the arterial system a state of spasm more or less permanent which rapidly produces hypertension, and consecutively arterio-sclerosis; but to him, judging from his earlier writings, arterio-sclerosis is a chronic inflammation of the small vessels, an endarteriolitis. He expressly states that "arteritis of the small vessels is, in short, the anatomical characteristic of arterio-sclerosis." Broadbent recognises peripheral contraction as a cause of heightened blood-pressure. Allbutt fully recognises the correctness of the general proposition.

Controversy has, curiously enough, been waged round the question as to whether or no there is heightened arterial tension without structural changes in the arteries; and this problem can be best approached by indicating the order in which phenomena appear.

The first step is the presence in the blood of toxic substances, or of "muscular excitants," to borrow an excellent term from Huchard. These cause hypertonic contraction not only of arterioles but of arteries, which have a muscular coat, at least up to the size of the brachial. The capillaries often share in the contraction. The presence of this hypertonic contraction is quite evident in the radial arteries, the temporals, and other arteries within reach of the finger. If the muscular excitants are continuously present in the blood the hypertonus is continuous, and the degree of the latter must be proportionate to the measure of the former. The effect of this tightening up of the arterial wall on blood-pressure has been already discussed; it leads to an increase of pressure in the aorta, and it does this without any increase in heart action; although an increase is called out, if there be any reserve, to overcome the greater difficulty of opening

the aortic cusps. That there is an equal raising of pressure in the constricted vessels themselves is an altogether different question. The constricted vessels offer greater resistance, so that less blood flows through them and less reaches the capillaries. The conception has been that the increase in pressure compensated for the constriction, and kept up the blood supply in the constricted area, whereas what really occurs is that the constriction throws the increase of pressure back on the non-constricted vessels and on the aorta. The arterial system being made up of sections, and the heart having a self-regulating mechanism, the increase of aortic pressure is determined by the extent to which constriction is general, and by the reserve of power in the heart. That arterial contraction raises blood-pressure by from 100 to 200 per cent. in the constricted vessels is the outcome of the belief that the hæmomanometer under all conditions only registers blood-pressure.

The recurrence or continuance of the hypertonic contraction leads to hypertrophy of the muscular coat of the arteries. That hypertrophy occurs has been shown by George Johnstone, Savill, and myself. That the degree of it is influenced by the measure of the blood-pressure is more than probable. Mere contraction of vessels does not lead to hypertrophy, but does so if long continued and if the blood-pressure be fairly maintained. But let me repeat that the maintenance of blood-pressure depends upon the power of the left ventricle. This explains the production of thickened vessels in vigorous people, and comparatively early in life. These are the people who, when they are seen early enough, have thickened and well-filled arteries, but in whom under appropriate treatment the thickening may disappear. If it does not disappear it is structural. When hypertonus persists it *must* lead to hypertrophy, hypertrophy being the normal result of such a condition. In my own observations, when the condition was permanently established, I found that not only was there this thickening of the media, but a thickening of the intima also, and sometimes of the adventitia in addition. The thickening of the intima is the result of a long-continued irritation of its tissue by substances present in the blood. Savill does not seem to have noted this intimal thickening in his cases; while the French

school is strong on intimal thickening, and says nothing about the media. Senator thinks that when arterio-sclerosis can be recognised clinically, it is extremely probable that all three coats are affected; but then he does not separate atheroma from his conception of arterio-sclerosis; and in atheroma all three coats are affected, but very differently to arterio-sclerosis. It seems to me that this failure to separate the two pathological conditions of atheroma and arterio-sclerosis has prevented clinicians realising the significance of processes constantly going on under their fingers and eyes.

The physiological corner-stone of my contention is that the vessels contract under the direct influence of irritating substances present in their contained blood. The same substances act upon the intima. The development of hypertrophic and of hyperplastic thickening is the result, and has its analogue in other situations.

The controversy as to whether a rise of blood-pressure precedes or follows vascular change becomes thus one of those circular arguments to which there is no end until the true steps of the process are determined. As in many morbid processes, the first step is an exaggeration of a normal process, and in the matter at present under consideration it is seen in tonus becoming hypertonus; the blood-pressure in the hypertonic arteries not being determined by the constriction, but by the state of tonus of other vessels, and by the power of the heart. There is no absolute sequence; for although hypertonus is the forerunner of sclerosis, sclerosis need not follow hypertonus. But I cannot imagine substances in the blood producing wide hyperplasia of the tunica intima and not causing prolonged hypertonic contraction of the media, and its consequential hypertrophy.

The term *presclerosis* is as undesirable when applied to the arteries as *prehypertrophy* would be applied to the heart. It belongs to the *pseudo* and *para* type of term, which is not regarded with scientific favour. The adoption of such terms tends to obscure truth, and even for the time to lull inquiry.

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CHAPTER IX

THE PRACTICAL APPLICATION OF THE PRECEDING CONCLUSIONS

VESSEL SENSITIVENESS AND THE CLINICAL SIGNIFICANCE OF HYPERTONUS.

THE INTERPRETATION OF HÆMOMANOMETER READINGS.

In the preceding chapters conclusions have been arrived at, the practical significance of which it is desirable to formulate at this stage, as it will make the teaching of the later chapters easier to follow.

First—I have defined my use of the term Hypertonus; and indicated the change in the arterial wall, and the diminution in the vessel lumen which characterise it.

Second—It has been shown that hypertonic contraction is caused by the presence of substances of various kinds in the blood.

Third—It has been argued that continued hypertonus leads to hypertrophy of the tunica media; that this is commonly associated with thickening of the other two coats; and that the term arterio-sclerosis ought to be confined to these changes.

Fourth—The relationship between vessel contraction and blood-pressure has been considered, and various errors have been dealt with.

Fifth—It has been indicated that sclerosed vessels retain their contractility, and are not the rigid tubes they are commonly described as being.

Sixth—The instruments used to measure blood-pressure only do so in normal vessels; in thickened vessels the readings are largely influenced by the arterial wall.

Seventh—As will be shown subsequently, in arterio-

sclerosis hæmomanometer readings rise with hypertonus of the sclerosed vessels, and fall with its reduction, but thick-walled arteries never give normal readings.

Eighth—The hæmomanometer can supply a record which, as will be apparent later, is not only of interest, but of clinical value, when properly interpreted.

The following chapters taken from my clinical observations are used to illustrate my various contentions; but before passing to them I desire to direct attention to what may be regarded as *vessel sensitiveness* under the following head:

VESSEL SENSITIVENESS AND THE CLINICAL SIGNIFICANCE OF HYPERTONUS.

It is probable that vessel sensitiveness varies greatly in individuals; judging, indeed, by the recognised differences in the tissues and organs generally of different persons, we are entitled to hold that like differences exist in the vascular walls. The recognition of this is of practical importance, for it leads us not to look at the vessels as a mere mechanical system of tubes, the pulse in which indicates the degree of heart power, but to form our estimate of the individual by recognising the state of his vessels. Looking at the patient with a true picture of his vascular system before our minds, our estimate will assuredly be more correct. In fact, no reliable estimate is possible without such a mental picture. A persistent hypertonus, for example, is abnormal, and if its presence is recognised it will lead us to appreciate symptoms which might otherwise be regarded as wholly fanciful. Such symptoms associated with vascular manifestation are often the first steps and the beginnings of processes which become permanent anatomical changes. Regrets are from time to time expressed that we do not know, do not see, the beginnings of morbid processes; that we only know them when fully established. Here, as in all other departments of life, we only see what we have eyes to see. The coarse, the sudden accidents which befall man necessarily first attract attention; the coarse results of disease long occupied men's minds: it is only now that we are in a

position to apprehend the subtle modifications which lead to manifestations which were deemed unimportant, if recognised at all, a relationship which it is now our privilege to recognise, at least in some degree, and to make some useful application of our knowledge.

The position in outline is this: the individual, with his physical and mental endowments, the expression of his heredity and environment, feeds himself. His tissues and organs are nourished by a nutrient fluid whose intimate and subtle composition is determined by heredity, by what he eats, and by the activity or efficiency of his excretory functions. Even these last may be largely hereditary, or, what is equivalent to it, congenital. The hereditary factor is the characteristic of the individual, his "constitution," his special and peculiar chemico-vital composition: to what extent it can be altered is still a much debated question. This factor, it may, however, be safely said, is more or less permanent; but its manifestations depend upon the pabulum supplied to it. The man who is congenitally what we call gouty will assuredly manifest his heredity under certain feeding conditions, and will not do so under other conditions. His heredity may remain in abeyance; whether it can be eliminated by prolonged abeyance, whether it might, so to say, disappear from never being called out, is a problem difficult to determine with rigid accuracy. In this connection it may, however, be remarked that people who once show idiosyncrasy do not, in my experience, lose it. Persons who get urticaria from eating shell-fish manifest their idiosyncracy right through life. An old friend of mine continued to be poisoned by egg in the smallest quantity up to the time of his death at four-score years; a lady, well on in middle life, lost in no degree the severity of the symptoms of spinal cord poisoning which infusion of ordinary tea induced in her. So throughout the whole mass of individuals there is this personal factor; and equally in the individuals with whom we have to deal this hereditary factor can be modified, can be helped, strengthened or restrained by the composition of the blood. Not that the blood-forming organs are altered, but their working is and must be modified by the blood supplied to them; while outside the indirect contributions from those

organs the blood depends directly upon the feeding. But the blood channels are not only the conduits for carrying nourishment, for supplying the raw material from which various elements and ingredients of the blood itself are manufactured; they are also the sewers of the body, taking away the waste from every cell and organ. The efficiency even of this function may be determined by heredity, for it depends upon the thoroughness of the metabolic and katabolic processes in the individual cell,—that is, upon its inherent vigour. We may not be able to greatly increase that vigour, but we can do much to prevent its being paralysed by an excessive call for work. If we understand the scheme right we can prevent the low-vigour cells being deluged with pabulum, which not only they cannot utilise, but which smothers the power they do possess. The medical problem circles round the three factors of hereditary character, pabulum, and excretion. Fortunately, the pabulum conduits, which it must be realised are also the sewage conduits, are not mere elastic tubes, but living tubes, responding to the composition of the contained blood, narrowing their calibre when the blood contains some hurtful substances, dilating when paralysed by other substances. That this property of direct response on the part of the vessel wall to the composition of the blood has for its object the protection of the tissues seems to me to be its true explanation; it takes away the purely physical conception of the circulation, and raises our conception of it to a level with our conception of other systems.

We thus come to the conclusion that sustained hypertonus indicates the continued presence of substances in the blood which act by irritating the vessel walls, leading to their hypertonic contraction. These substances betray their hurtfulness by their action, for sustained hypertonus is hurtful: it raises peripheral resistance and keeps it unduly high; it prevents the peripheral flushing with blood which is required for full cell vigour, and for the complete removal of refuse and waste.

This is a large generalisation, and yet it is no sooner put into words than its truth is apparent. The knowledge we already possess warrants us in regarding this as a fact—

as a law—as the fundamental fact on which a correct conception of the relationship between metabolism and the circulation is to be based. It becomes a new guide to us, an indicator of the more subtle changes taking place in the fluid upon which health and vigour depend.

This is not only true of normal vessels, it is true of sclerosed vessels; *their tightening up indicates the presence of deleterious substances in the blood*, they relax as these are removed. The proposition that sclerosed vessels can be more sensitive than normal vessels to such influences has been a difficulty with some of my friends, yet it has been shown by the physiologists that vessels become more sensitive, acquire an "exaggerated irritability," to some of the constrictor substances, referred to in previous chapters, after degenerative section of the vasomotor nerves. It seems to me that in the sclerosed vessels of old people this extreme sensitiveness is sometimes very marked, and although, as I have stated in Chapter IV. page 29, there are certain possible fallacies, there is no doubt as to the existence of this extreme sensitiveness, and that it persists after nerve impulses have clearly become dulled. The recognition of this is of immense importance, for arteriosclerosis may be unaccompanied by symptoms requiring medical skill, whereas the advent of hypertonus always heralds symptoms referable to one or other organ. The hypertonus becomes thus an indicator of blood condition, not of nerve-centre perturbation. The brain perturbation, when present, is of vascular origin.

THE INTERPRETATION OF HÆMOMANOMETER READINGS: ANGIOHÆMOMANOMETER OR ANGIOMANOMETER.

From what has been said in previous pages it will have become apparent that my contentions lead to the conclusion that the two factors in the determination of *arterial pressure* or *compressibility*, as measured by the instruments in use, are (1) *blood-pressure*, and (2) *the thickness of the wall and the proportion it bears to the lumen.*

When observations are made on persons with soft and absolutely unthickened vessels the arterial pressure ap-

parently corresponds with the pressure that would be obtained were a cannula introduced into the artery. In such persons there is a rise and fall of pressure equal to about 20 mm. Hg. in the 24 hours. I do not doubt that there is this measure of variation in true blood-pressure. I do not question the proposition that the instrumental readings record this, and that in the normal daily variations the state of the vessel wall may be negligible; and yet it will commonly be found that the higher readings coincide and correspond with some hypertonic contraction of the vessel wall. As soon, indeed, as readings go above normal, it will, in the vast majority of instances, be found that they are accompanied by a definite thickening of the radial wall, and that the fall in pressure coincides with the softening and relaxation of the artery, and corresponds with it.

The artery we depend upon for our finger observations is the radial, while the vessel compressed is the brachial; and it is well to bear in mind that the condition of the wall of the brachial is not always duplicated in the radial, or *vice versa*; in the great majority of instances it is so, but one meets with exceptions on both sides, sufficiently few, however, to prove that correspondence is the rule.

When the radial is thickened from structural changes the pressure remains constantly above normal; the degree of thickening and its proportion to the size of the lumen determining the height of the instrumental reading. When a thick vessel tightens up in hypertonus the reading increases by from 20 to 40 mm. Hg. or more, and falls as it is relaxed.

The fact that, with a thick vessel wall, the reading often never falls below say 200 mm. Hg., no matter how poor the power of the wave inside the vessel may be, and no matter what means be taken for the reduction of the reading, is really an absolute proof that the vessel wall takes a large share in the resistance to the compressing bag. That being so, it necessarily follows that the vessel wall, save in the perfectly normal vessel which is only open when it is full of blood, has to be reckoned with. The contrary contention has entirely discredited the use of the hæmomanometer with many accomplished clinical pathologists, and the result is not to be wondered at.

HÆMOMANOMETER READINGS

When it is recognised that the arterial wall is such an important factor it will be found that some moderately high pressures, say 140 to 150 mm. Hg., are due to hypertonic thickening, and that both pressure reading and thickness of wall can be easily reduced to normal; that in other instances still higher pressures, say 220 to 240 mm. Hg., can never be reduced to normal, but that a lowering of 20 to 40 mm. Hg., or rather more, may be induced, and that it corresponds with a measure of wall relaxation which is readily appreciable to the finger. The fall is the result of the relief of the hypertonus. I have already said it is the hypertonus which is the essential phenomenon; and as hypertonic contraction still further thickens the wall and reduces the lumen of a sclerosed vessel, the reading must be raised under the circumstances, and will fall with the disappearance of the hypertonus. I do not believe that any who have the abundant opportunities of carefully watching thick vessels that I have, will have any serious difficulty in satisfying themselves as to the correctness of these statements. I must, however, again add a warning note to the effect that feeling the radial is not always a reliable guide as to what the brachial pressure is to read. I have already referred to this, and I shall only add that in some cases the radial artery and its pulse would not lead one to suppose that the brachial pressure would be high. I have two such cases under observation as I write this, the radial artery being neither hard nor incompressible, and yet in both there is a steady reading from the brachial of over 200 mm. Hg. In another case, also under present observation, in whom frequently repeated doses of erythrol dilated the capillaries so that the patient became ruddy in appearance instead of pale and haggard looking, and yet his brachial pressure, which was over 200. was not reduced. On the other hand, the brachial pressure may be lower than the state of the radial suggests. I have seen calcareous radials the pulse in which was stopped by a brachial pressure of 200, while had the brachial been like the radial its wall could only have been compressed by splintering it. One other reservation has to be made, namely this, that occasionally a pressure of 110 mm. Hg. will become 120 by relaxing the vessels—

the wave in the radial becoming larger and more vigorous. I have not seen many instances of this, and at first they were perplexing, but the explanation seemed to me to be clear, namely, that the relief of hypertonus freed the heart and allowed it to act with so much greater vigour that the loss from the change in the vessel wall was more than balanced by a true rise in blood-pressure.

I mention these apparent exceptions to the propositions I have submitted, for I believe that their occurrence has misled observers. I acknowledge the difficulty they presented to myself, but steady and close observation in due time solved the riddle, so far as I was concerned.

Were I ambitious to add another word to the rich new vocabulary in medicine, I would suggest *angiohæmomanometer* as a further development of the word hæmomanometer, or *angiomanometer* as a satisfactory substitute for hæmomanometer.

REFERENCE.

Elliott, *loc. cit.*

CHAPTER X

HYPERTONUS AS SHOWN BY THE SPHYGMOGRAPH

THE following four cases are representative of classes of cases. They all showed hypertonic thickening of the radial arteries when they came under observation, and they were all treated by anti-spasmodics with a view to the relief of arterial hypertonus. The results of the treatment on the arteries are shown in the sphygmographic tracings.

CASE 2.—A man, aged 35 years, came to me at the Edinburgh Royal Infirmary complaining of headache and general weakness and insomnia. He was a strongly-built, well-nourished man, but was somewhat pale. He had been working at his present occupation for three months, and before that time had been well. The radial artery was markedly thick, the thickening being uniform and extending right up the arm. In fact, it presented the characters which would commonly be described as "arterio-sclerotic." The man was young, and this, combined with his symptoms and the absence of any evidence in the urine of renal disease, led me to surmise that this vessel condition was largely one of hypertonus and that, even assuming that there might be some degree of thickening, his complaints were due to the hypertonus and to the cause, whatever it might be, producing it. A sphygmographic tracing was taken, and he was admitted into Ward 23. Some doses of erythro-tetranitrate demonstrated that hypertonus was accountable for most, but not all, of the radial thickening; this drug, while relaxing arterial spasm, does not, of course, remove an organised thickening. In this case the thickening was greatly reduced

by relaxing the spasm. The sphygmographic tracing may be compared with the former one. The existence of hypertonus

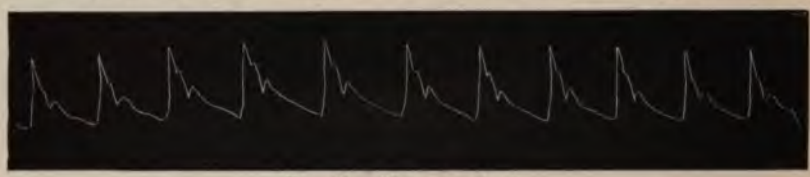

FIG. 21.—Before erythrol.

FIG. 22.—After erythrol.

in this case was of special interest, as we found afterwards that the man had had syphilis.

CASE 3.—The following tracings were also from a patient in Ward 23 suffering from spasmodic asthma. He was a man, aged 53 years, and it was quite clear that when the

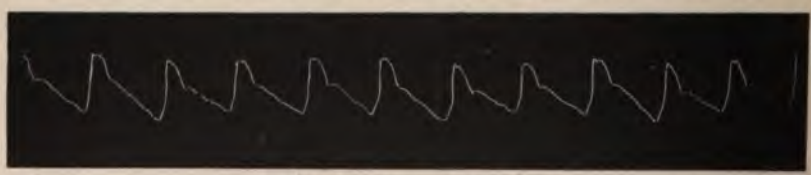

FIG. 23.—Before liquor trinitrini.

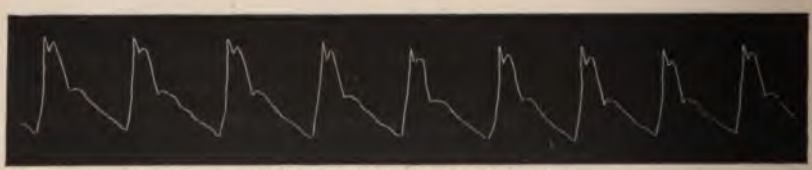

FIG. 24.—After liquor trinitrini.

asthmatic spasm supervened his radial arteries became markedly tightened up. The following are the pulse tracings during the asthmatic spasm, and after its relief by means of liquor trinitrini.

CASE 4.—These tracings were from a man, aged about 60 years, also in Ward 23, suffering from extreme orthopnœa, due to myocardial degeneration. The vessels were thickened,

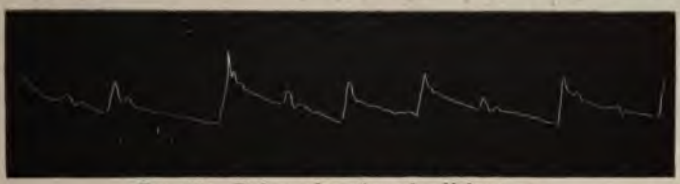

FIG. 25.—Before relaxation of radial artery.

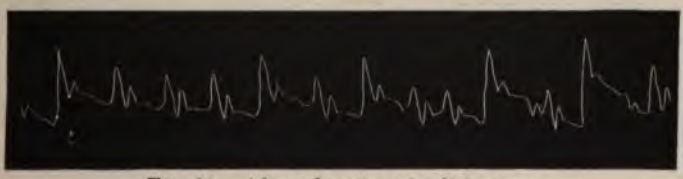

FIG. 26.—After relaxation of radial artery.

but they also seemed to me to be in a condition of hypertonus. He got great relief from the administration of anti-spasmodics which relaxed his vessels.

CASE 5.—The next tracings were from an old man, aged 70 years, who was under my observation for years in Queens-

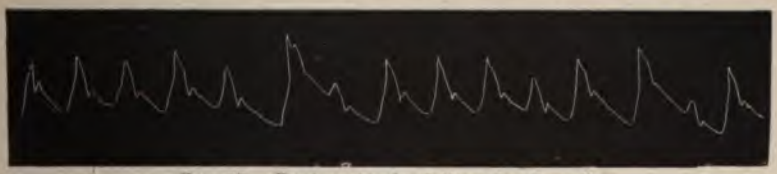

FIG. 27.—During attack of "renal asthma."

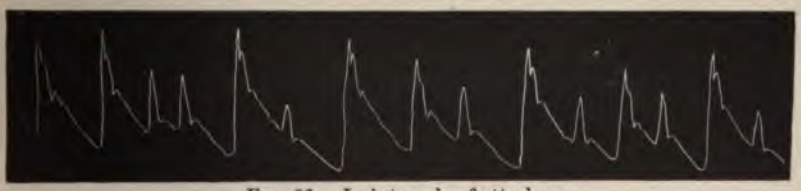

FIG. 28.—In intervals of attacks.

berry House. He was subject in his later years to recurring attacks of "renal asthma," during which his permanently thickened vessels quite appreciably tightened up and became

thicker. The sphygmograph gave the following tracings during the attacks and in the intervals.

In these four cases the difference in the pulse when tightened and not tightened was quite appreciable to the finger. The sphygmograms entirely bear this out. Under hypertonus, with the well-filled thick-walled artery, the swing of the lever is less, the percussion stroke is shorter and less abrupt, the summit tends to be more rounded, and the predicrotic notch less evident. When the hypertonus passes off all this is altered.

In the chapters which follow I endeavour to show, firstly, the association of hypertonus with sclerosis; secondly, the significance of manometer readings in a number of abnormal conditions; and how when correctly interpreted the records are both interesting and helpful in everyday practice.

CHAPTER XI

THE CLINICAL SIGNIFICANCE AND VALUE OF HÆMOMANOMETER OBSERVATIONS IN ADVANCED INTERSTITIAL NEPHRITIS

THE condition to which we instinctively turn in the first place for the clinical proof of the accuracy of the contention as to the influence of the arterial wall in high hæmomanometer readings is advanced interstitial nephritis. In it the vessel changes, which are very marked, are universally recognised. Such vessels as the radials and temporals, to which clinical observation is commonly confined, are extraordinarily thick and hard, and compared to whip-cord. The pulse in such vessels is, as has been already mentioned, usually described as a "high-tension pulse," as an "incompressible pulse," or as a "hard pulse." Medical literature is full of these terms, and there is abundant evidence available to prove that what was meant by them was really blood-pressure. The conception was that there was peripheral resistance, that to overcome this the heart worked with increased vigour, the consequence of which was heightened pressure inside the vessels, and from this arose the conception of "high-tension" pulses. Blood-pressure measuring instruments have been used to illustrate and to support this view, so we propose to follow the same method of investigation, with a view to ascertaining the significance of the records these instruments give us.

As a preliminary to this I present in Figs. 29 and 30 sections of the radial artery taken from two cases, both fatal, from large cerebral hæmorrhage. Fig. 29 was taken from a young man of 28 years, who was known to have subacute interstitial nephritis, and whose radial artery was thick and hard. After a day or two of energetic treatment for uræmic

in extremis from uræmic dyspnœa with acute engorgement of the lungs. She had been under treatment on one or two occasions for some eye condition. On admission to the medical wards her condition was as follows. The radial arteries were very hard and thick, the heart was enlarged and thumping; the urine contained albumin, casts, and some red blood cells. The urea was 4 grains per ounce. There was no œdema. In June the hæmomanometer gave a reading of 280 to 290 on the only occasion I at that date used the instrument for her.

This patient was in hospital for months, the rough history of her condition being that from time to time she threatened to become uræmic, and was actively treated whenever the symptoms manifested themselves. The "blood-pressure" taken on different days and at different hours during September are given below to illustrate the variations, while some remarks are added.

			Mm. Hg.	
Sept.	10.	Forenoon . (Oliver)	260	Constipated
„	12.	4.30 p.m.	250	Bowels have been freely moved.
		9.30 „	255	Nothing special to note regarding her condition.
„	13.	1.30 a.m.	245	
		1 p.m.	260	
		3 „	250	
		7.15 p.m.	255	
„	14.	Midnight	245	
„	15.	Noon	270	
„	21.	10 p.m.	265	Uræmic since 18th; been treated by hot pack, etc.
		„ „	250	After hot pack.
„	22.	8.30 p.m.	255	
„	23.	8.30 „	255	
		11 p.m.	245	
		Midnight	245	Uræmic symptoms subsided, condition much improved.
„	24.	8.30 p.m.	230	
„	26.	1 p.m.	240	

The pressures in this patient are thus shown to have varied between 270 and 230,—below the latter figure pressure did not fall. It was noted in this case that if the bowels were not moved the pressure rose, while after being moved there would be a fall, as seen on the 10th and 12th, when it fell from 260 to 250. The effect of hot packs in

reducing the pressure is also shown. When symptoms of uræmia began to show themselves there was a quite definitely palpable increase in the thickening of the radial artery from increased contraction of it. This is the change commonly spoken of as increased "tension," and is thought of as blood-pressure, when it ought to be regarded as vessel tightening up. With this increase of contraction and of consequent thickening the hæmomanometer always gave a higher reading, while as the vessel relaxed the reading fell. In this class of case, with a hypertrophied and strongly acting heart and the blood well filling the vessel, the use of the term "raised tension" is not unreasonable; the great drawback to it is that it has led to the essential fact being obscured, for it has not been recognised that the tightening up of the vessel itself is the important and essential part of the phenomena. The measure of increase in the intra-arterial pressure, which is, of course, the only *true* blood-pressure, has been not only much overrated, but it does not even necessarily occur.

CASE 7.—Mr. S., aged 62, was sent to me from the North of Scotland, and his condition was such that I advised him to go into the Royal Infirmary. He was admitted on the 10th September. He complained of his eyesight having failed greatly, and on examination he was found to have extensive albuminuric retinitis. The radial arteries were very thick and hard; the heart was enlarged, and its impulse forcible. The urine contained albumin and casts. He had morning vomiting. The hæmomanometer gave a record of 260. After some improvement under careful dieting and medicinal treatment he became steadily worse, and died on the 15th, five days after admission.

The following observations were made on this patient's arterial pressure:—

		Mm. Hg.	
Sept. 12.	4.30 p.m.	(Oliver) 270	
	9.40 „	245	
„ 13.	11 a.m.	220	
	1 p.m.	245	
	3 „	240	
	7.15 p.m.	260	
„ 14.	Noon	265	
	8 p.m.	300+	
„ 15	Noon	295+	Died in afternoon.

In this patient, while the hæmomanometer gave pressures which fell from 270 to 220 as the result of treatment, this latter figure was the irreducible minimum. The variations in the readings could be appreciated by the finger,—the radial definitely relaxing as the pressure fell. The unfavourable course was characterised by increasing constriction of the radials, and a corresponding rise in hæmomanometer reading until the heart failed. The changes in the radial artery were unmistakable to the educated finger. The conclusion that in the final stage of this disease the vigour of the heart can rise to something like 200 per cent. above the normal is unavoidable if the readings of our instruments are to be taken as representing *blood-pressure*. To me the proposition that with the failing heart in this patient the blood-pressure was rising seems little short of grotesque. Whereas the increasing tightening up of radial arteries—the wall thickening and the lumen diminishing—gives a reasonable explanation of the instrumental readings; and these alterations were unmistakably to be felt by the finger. They can even be seen when the vessel is superficial in position.

Case 8.—Mr. M., aged 40, was sent to see me about the middle of July 1906. He had only been conscious of not feeling well since about the beginning of the year. It was known that he had albuminuria, and the question was whether he was to be allowed away from home for a holiday. When I saw him the radial arteries were very thick and hard; the pulse was frequent, beating over 100 per minute, while the wave was small and feeble. The heart was enlarged, and the first sound was faint. I advised that he should go home, go to bed, and be carefully watched and treated by his medical attendant. The prognosis I considered was extremely gloomy. After being kept in bed till about the end of July, I was asked to see him again. He had improved considerably, and I was sanguine that the improvement would continue and make further advance. This seems to have been the case for some time, but he somewhat suddenly became worse, and died after a convulsive seizure. The second time I saw him the hæmomanometer gave a reading of 250 mm., which was the only observation made.

This is a type of case one sees from time to time in the consulting-room and in hospital. They may or may not have been under skilled observation, symptoms having sometimes been disregarded. In such cases it will be found that the higher the pressure reading exceeds 200 the nearer is the inevitable end. It means a greatly thickened artery, and a tightly constricted one,—the former a permanent anatomical thickening, the latter a hypertonic contraction. The latter factor is due to the blood, loaded with nitrogenous waste products, so irritating the vessel wall as to lead to its contraction, just as we have already seen that digitalis acts.

In fact, in this disease we have a demonstration of the contention that "waste products" in the blood act upon the vessels by constricting them. This is a recognised fact although there is a curious hesitancy in fully applying it. However ignorant we may be of the precise nature of the "waste products," the state of the kidney leaves it unquestioned that normal depuration is grievously hindered.

CASE 9.—Mrs. M., aged 37, was admitted to the Royal Infirmary on 22nd September. The radial arteries were thick and hard; there was albuminuria, and both eyes showed marked albuminuric retinitis. The highest manometer reading I have of her artery is 235 mm. Hg. She stayed in hospital for a few days only, as she wanted to return to her family.

The following observations were made in this patient:—

			Mm. Hg.	
Sept. 22.	8.30 p.m.	(Oliver)	220	
„ 23.	8.25 „		230	
	11 „		200	After magnes. sulph. had acted freely.
	Midnight		205	
„ 24.	8.30 p.m.		210	
„ 26.	1 „		235	

In the preceding three cases in which a number of observations were made, the readings varied in

Case 6 . . . from 230 to 270 mm. Hg.
Case 7 . . . „ 270 to 300+ do.
Case 9 . . . „ 200 to 235 do.

while in Case 8, with one observation it was 260 do.

If the normal be taken as 100 to 120—the precise figure not being of much importance for our present purpose —we have here four cases with an increase of from 100 per cent. upwards—two, two and a half times, almost three times the normal. And we are asked to accept this as "blood-pressure"! The claim, if it means anything, is this,—owing to conditions in the blood the peripheral resistance is increased, and the heart, responding to the call for more work, increases in power so as to cope with the resistance, and develops a power which gives even in the brachial artery a pressure from say twice to two and a half times more than the normal. With the fullest appreciation of the reserve power in the heart we cannot but think there is a big error here. Taking Case 6 as the case in which the greatest number of observations were made, it is seen that the readings ranged from 230 to 270, a margin of 40, but below this it never fell; in Case 2 it never fell below 220; and in Case 3 never below 200. The lower figures were in some instances the direct result of treatment, but no kind of treatment reduced them further,—this apparently was the irreducible minimum. In all four cases the disease was the same, and in all there was great and permanent thickening of the arterial walls. Now what is done by such instruments as we use is this—the pressure in them is raised until the arterial circulation is arrested in the part; this means that the artery is so compressed by the pressure of the surrounding soft tissues that its lumen is obliterated. The result depends, therefore, upon the compressibility of the artery, and the compressibility of a tube, as has been already shown, depends upon the thickness of its wall. The question has been dealt with from the purely physical side, and it is seen that the clinical facts, in the condition dealt with in this chapter, are in complete harmony with them, and indeed with common knowledge as well.

In our four cases there was no doubt about the great thickening of the arteries; and this must be regarded as having been a large factor in determining the high manometer readings. The *degree* of thickness of the arterial wall becomes therefore a factor of high value in determining the *height* of hæmomanometer readings. The cases gave further

proof of the accuracy of this. It was quite unmistakably observed by the finger that when the manometer readings fell the radial artery became slightly softer and larger. This was corroborated by others, for when attention was directed to the changes in the radial artery I found that my assistants and others soon acquired the faculty of recognising the changes, and soon became much interested in their observation. On the other hand, when the vessel tightened up the manometer reading rose. The relaxation of the radials soon after the administration of erythrol I have demonstrated to a class of graduates, and shown that it corresponded with a fall in manometer reading. In the same way a hot pack relaxes the radial artery so as to be apparent to the sense of touch, while the manometer reading was reduced; so also, after purgation or free bowel movement, there was relaxation and lowered reading. Instead of recognising and watching the vessel changes in this disease, clinicians have thought only of the blood-pressure in the radial artery; and I do not doubt that the increased thickening or hardening of wall was usually called tension, and thought of as blood-pressure.

The determination of how much is blood-pressure, and how much is to be attributed to the increased thickness in the vessel wall, does not seem to me to be capable of instrumental proof. The lowest record in these four cases was 200 mm. Hg. and no matter what measures were taken this great heightening in the readings persisted, just as the thickening of the arterial wall persisted, and to which I contend it was due. The readings, however, rose and fell. In Case 6 a pressure of 230 would rise to 270, a rise of 40 mm. Hg., a greater rise than the physiologists tell us takes place after violent exertion; and we are asked to believe that a heart already doing 100 per cent. more work than normal finds no difficulty in doing this extra 40 mm. Hg. In Case 7 the pressure rose to over 300 mm. Hg. when the patient was dying and the heart giving out. I submit that when these rises occur the radial artery contracts, that as a consequence its wall still further thickens and its lumen diminishes; and, seeing that the arterial wall takes a large share in giving the high manometer readings, this hypertonic contraction of the sclerosed vessel explains the further increase in the manometer reading. The hyper-

tonic contraction, as I have already said, can be followed by the finger, so that there is no doubt whatever as to its taking place. What the blood-pressure is inside that tightened-up artery is a totally different problem, and one which can only be dealt with and investigated indirectly. I submit that **the readings do not represent blood-pressure.** That the state of the arterial wall is sufficient to explain them is probably not far from the precise truth. In fact, my belief is that not only in chronic kidney disease, but in all disorders, the arterial wall has to be given a prominent place when hæmomanometer readings are interpreted. That being so, vessel thickening, whenever present, takes from hæmomanometer readings the value which has been attributed to them as records of real blood-pressure.

At the same time, it must be equally insisted upon that these records are of interest and of value, and are not to be put aside as worthless,—the fate which has befallen them at the hands of not a few very able and critical physicians.

With arteries such as are invariably met with in advanced interstitial nephritis high readings will always be got; but, as has been shown in the preceding cases, there is a wide range of movement in the readings, according to the condition of the patient, although the high level is always maintained. This range indicates the margin within which remedies act, and the effect of these will be found to be usefully defined and represented by the readings. Had I to make the choice I would select to have my fingers rather than the hæmomanometer; but I am glad to have both, for the instrument gives me a record of numbers which I can present to others, while the skill of my fingers belongs entirely to myself, and cannot be transferred to a chart. The instrument, in such cases as those recorded, gives a minute record from hour to hour, and day to day, of the state of the arteries; and as my contention is that the varying degree of arterial contraction depends upon the condition of the blood, the readings indicate that condition, and they thus become an invaluable guide to treatment, and to the daily management of these critical cases. It is desirable, however, to accentuate the point that a high pressure is not necessarily an indication that relaxing and reducing treatment is to be adopted. In

Case 9 a pressure of 235 could stand active measures for reduction; in Case 6, with the pressure reduced to 230, it would have been unsafe to have continued the relaxing treatment longer; while in Case 7 I was not satisfied that the pressure, when lowered to 220, had not somewhat overstepped the safe limit. It is here the skill of the physician's finger comes in,—it is by that means that the wave inside the hard vessel is estimated, and no instrument can dò this for us, nor, I venture to predict, will an instrument ever be devised to replace the *tactus eruditus.*

CHAPTER XII

THE INFLUENCE OF THE ALIMENTARY SYSTEM IN CAUSING ARTERIAL HYPERTONUS AND SCLEROSIS: FOOD, DIGESTION, CONSTIPATION, AND ALCOHOL.

THE INFLUENCE OF DIET ON SCLEROSED VESSELS.
THE INFLUENCE OF DIET ON THE ADRENALS.

It has been long and fully recognised that certain conditions having their origin in the alimentary tract were accompanied by changes in the pulse. The changes have come to be generally referred to as alterations in blood-pressure; alterations which modified or changed the relation between the vessel wall and the circulating blood within it. The terms in common use to define the change have been "incompressibility" and "tension"; terms indicating either the conception formed of the strength or force of the stream, or the state of the vessel wall as determined by the blood inside it. That these terms have come to be very inaccurately used by many practitioners will not be disputed. Controversy has taken place as to the meaning of the word "tension," and, so far as I am able to judge, the word as used in clinical medicine has not the same significance as it has to the pure physicist. The word has been borrowed from physics, where it has a very wide significance, and applied in clinical medicine to indicate one set of phenomena. To the purist the use of the word in medicine tends therefore to be either an offence or a stumbling-block. It seems to me that in clinical medicine it is used to express the mechanical effect of the circulating blood and the pulse wave upon the vessel wall as it can be estimated by the educated finger. The effect depends of course upon the ful-

ness of the vessel. If the vessel continues well filled during diastole, the wave which travels along it with each cardiac systole gives to the finger the feeling of greater fulness, with a sense of obstruction in the vessel wall to the stretching.

It is these two factors which are comprised in the word "tension" as intelligently interpreted. The feeling of greater fulness, which may be present even during diastole, is produced in the radial artery in two ways—namely, by a constriction of the vessels on the distal side of the wrist, or by a contraction of the radial artery itself.

This sense of increased fulness is due in the first of these to a local increase in the amount of blood in the vessel and to a local rise of blood-pressure in the radial artery. No change in the action of the heart is necessary for the production of this effect; it is the inevitable effect, and it is a purely physical phenomenon. It can be felt in the radial artery by keeping one finger on it when the vessel is obliterated by another placed on the distal side of the point selected. It can also be shown by compressing the *superficialis volæ* branch while a finger is on the radial at the wrist. In both these simple procedures the radial artery is felt to become fuller and therefore tenser. The same feeling of *fulness* may be produced by the radial artery participating, as it commonly does, in capillary contraction. The contraction thickens the arterial wall and brings it into a different relation to its contained blood. As Broadbent long ago pointed out, the normal artery during diastole is oval when viewed in transverse section, while the passage of the pulse wave alters the oval to a round. When an artery is hypertonically contracted it changes from the oval towards the round, until with sufficient thickness it maintains the round outline. It follows that when the pulse wave passes along the hypertonic artery it gives through the finger a sense of greater resistance. I venture to believe that this was the "heightened tension" of our best clinicians. That confusion should have arisen between clinical observation and physiological teaching was inevitable. To the initiated, *tension* meant this relation between the blood and the vessel wall; and it was the true appreciation of this relation which revealed the *tactus eruditus* of the physician.

The idea of "fulness" in an artery is thus easy of interpretation, and is used in quite a sound sense; but it has given rise to another conception—namely, that there is an increased volume of blood—not a local but a general increase. This is not by any means so sound a view, although common enough; and it is one of the views which has shared not a little in hindering a more correct understanding of the phenomena presented by the circulation in a great variety of general disorders. The idea of increased volume of blood is of course the "plethora" of the past century, not to go farther back; while the idea of "depletion" as the remedy for "plethora" has survived, although transferred from blood-letting to purgation.

This fulness of blood, or full-bloodedness, was commonly associated with the idea of high, or large, feeding, and the free use of alcoholic liquors. It naturally enough was literally interpreted as the result of a too abundant supply of nutriment added to the blood. This type was seen in the person of florid face, bulky frame, and full, strong, and hard pulse.

The effect of the condition of the alimentary tract upon the pulse was, however, also recognised in another type. The individual in this class did not so plainly show that he indulged to excess, but yet he too suffered from attacks of "biliousness," or of "disordered liver." In him also the pulse was of "high tension"—it was harder, firmer, and less compressible.

It is indeed evident that a relationship between the alimentary tract and the circulation has been long recognised in medicine. The association of the two is no novel doctrine, for it forms now and has formed for generations the basis of treatment. The treatment has consisted of free unloading of the alimentary tract, and a reduction in the amount of and often an alteration in the kind of food and fluid usually taken. The result was the relief of the patient's symptoms, while the reduction of *pulse tension* gave countenance to the idea of depletion.

This is the common knowledge of the profession, yet to get the full meaning and the full value out of this commonplace belief and proceeding, it is necessary to examine the matter more fully.

INFLUENCE OF THE ALIMENTARY SYSTEM

We may assume that it is generally accepted that generous feeding and the free use of alcoholic and malt liquors lead to the condition of pulse which is called "high tension," especially if there be not daily, full, and free evacuation of intestinal contents. This is the gross type, and I think it is more common in some parts of this island than in others; and it occurs without doubt more commonly in some classes than in others. The individual of this type often dies in comparatively early life as a result of vascular changes in brain and kidneys. This gross type is readily accepted with all its clinical and pathological significance. It is accepted that liberal feeding and drinking lead to "raised arterial tension," to permanent vascular changes, and to early death the result of these. While this is the case, there is by no means the same frank recognition that there are necessarily minor degrees of the same class of phenomena, and that the minor degrees can produce similar results although taking a longer time to effect them.

I take it that it was the gross type that determined the idea of plethora, and its treatment by depletion; and that this has prevented the learning of the lessons which the lesser degrees were capable of teaching. In the gross type the idea was a heightened tension from over-filling—the vessels were too full, and the consequent pressure upon their walls increased the risk of rupture.

The more modern mode of expression is that "high tension" is due to "peripheral resistance," and yet this peripheral resistance seems to have an elusive existence when an attempt is made to come to close quarters with it. I do not question its recognition by some, but there seems to be often a vagueness about it which is most undesirable on such a very practical matter, and indicates a want of clear thinking which is equally undesirable.

There is no one more entitled to define the position than Sir William Broadbent, who rightly ranked as one of our greatest clinicians. He regarded capillary resistance as the cause of high tension; and amongst the causes of capillary resistance he included nitrogenous waste, and the products of imperfect metabolism. He also knew that capillary resistance might be due to contraction of the capillaries. This

seems to me to be as far as he got. The term *tension* seems to have satisfied clinicians, and therefore to have lulled inquiry; the introduction of the term *hypertension* is the latest corroboration of this suggestion.

It seems to me that the idea of "tension" has so dominated clinical medicine that the great and fundamental fact in the phenomena—namely, the fact of arterial contraction as it occurs in the radial and other arteries—has been entirely overlooked. This contraction, which I have called hypertonic contraction, has been considered in Chapters I. and IV., and it is unnecessary to repeat what has already been written. Here it is only necessary to emphasise the fact that the "high tension" of the "bilious attack," of the "disordered liver," of constipation, is determined by vessel constriction, and that that constriction is in the systemic arteries. That the capillaries are contracted also is certain, but whether as part of the initial contraction, or as merely secondary to arterial contraction, it is difficult to determine with certainty. At the same time, I distinctly lean to the view that the initial contraction affects both capillaries and arteries, my grounds for the opinion being the conviction that in some at least of the gastro-intestinal conditions which lead to "high tension," the vessel contraction is caused by the direct influence on the vessel wall of substances absorbed from the intestine, acting as digitalis, ergot, and adrenalin act, and that the capillaries cannot be excluded from such influences or from such action.

The question of the precise relations of the alimentary conditions, which are the primary cause of the vessel phenomena, is somewhat complex and requires detailed examination.

During the process of digestion there is a normal reflex, acting from the splanchnic area through the vasomotor centre in the medulla, which leads to systemic arterial contraction. This, however, is only one aspect of the influence of the alimentary system upon the blood-vessels; the further effect may be produced by two factors—namely, the absorption of an excess of the nutritive products of digestion, or the absorption of the products of intestinal putrefaction.

Taking the first of these two causes, it is not open to question that the big feeder presents the phenomena already

indicated. He eats more than his tissues require for repairing loss or maintaining energy, and, if he has a vigorous digestion, the conversion of his food into suitable nutritive pabulum is equally in excess, and his blood becomes surcharged with materials which cannot be utilised, which have to be disintegrated to make their removal possible. This extra work interferes with the metabolic processes necessary for the removal of the normal waste of tissue. In this way are supplied the "nitrogenous waste and the products of imperfect metabolism" which lead to the "bilious attack" with its vessel contraction and high blood-pressure.

The second factor to be dealt with is the absorption of putrefactive products from the intestine.

In the case of animal food, which embraces flesh of all kinds and eggs, it will be found that its retention in the intestinal tract leads to putrefactive changes. This putrefaction is brought about by the bacilli belonging to the colon group, which are the normal occupants of this tract. The process of putrefaction occurs most readily in the organic compounds present in animal albumin. In fact, for clinical and practical purposes, intestinal putrefaction may be regarded as always due to the decomposition of animal food. There is an important exception to this—namely, milk. This putrefactive decomposition of animal organic substances occurs in constipation, and, of course, the necessary sequence is that constipation being present, the products of decomposition are not got rid of by the bowel, or are only got rid of in direct ratio to the measure of intestinal evacuation. Constipation thus leads not only to putrefaction, but to the retention in, or absorption from, the intestines of the products produced during putrefaction. In the breaking up or cleavage of the proteid molecule in the putrefactive process, a number of substances are produced, and are absorbed from the intestine. Some of these substances are known to have a toxic and injurious effect upon the body. They all belong to what is technically known as the aromatic series. The best known of them are skatol, indol, and phenol. These aromatic products of intestinal putrefaction reach the liver, where they combine with sulphuric acid and are excreted in the urine as ethereal sulphates. The presence of an excess of

ethereal sulphates in the urine becomes thus the measure and gauge of the degree of intestinal putrefaction present. The measure of indican in the urine has the same significance. The indol is rapidly absorbed from the intestinal tract and carried by the portal blood to the liver, where it enters into loose combination with the liver cells. From this combination it is readily detached, to become united with sulphuric acid. Before becoming thus united, it becomes oxidised into indoxyl, so that when united it becomes chemically a potassium salt known as indoxyl sulphate of potassium. This substance is much less toxic than indol, finds its way into the blood, and is promptly excreted in the urine and known as indican.

In regard to the toxic properties of the substances referred to, there appears to be abundant room for further investigation into their action. The difficulties attending such investigations are necessarily very great, for it is to be remembered that in the human subject—and it is in relation to practical medicine that these questions really interest us—the toxæmia is often a slow and long-continued condition, the very slowness of which is apt to arouse scepticism in the minds of those who have not looked into the more subtle processes carried on in the animal body. Such an authority as Herter is satisfied of the toxic properties possessed by indol; that, in addition to its power to produce evanescent conditions as headache, irritability, and restlessness, it can, by its prolonged absorption, contribute to the production of chronic nervous disorder.

It may be asked, how do these effects compare with effects following upon analogous processes in carbohydrate food? And the answer is that while the fermentative processes which occur in them may lead to unpleasant symptoms, yet the substances produced are by no means so toxic or so injurious as those produced during proteid putrefaction.

While constipation is such an important factor in favouring putrefactive changes in the proteid contents of the intestine, it is necessary to state that other factors operate or co-operate in favouring these: defects in proteid digestion, due to disorder of gastric or pancreatic function, may lead to very imperfect and incomplete changes in the proteid food

taken, and such incomplete transformation favours the onset of putrefactive changes. A further important side to this question is that a diet, very rich in proteid, may be thoroughly digested, may be thoroughly converted into albumoses and peptones and ready for intestinal absorption, but the absorption may be prevented or delayed in consequence of their quantity or abundance in the intestine. This delay in absorption is fatal to those highly evolved products, for the organisms which produce putrefaction find in them material on which they act with great facility. There is thus a considerable variety in the conditions under which intestinal putrefaction occurs. In all there is the common factor of absorption of the products of putrefaction.

When there is this absorption of putrefaction products from the intestinal tract, there is, in the great majority of cases, the vessel contraction with which we are dealing—this is the main objective evidence supplied by the examination of the circulatory system, and, when present, the hæmomanometer reading is high, that is to say, high as compared with what it records when the condition is removed and the vessel contraction relaxed.

The symptoms and degree of discomfort caused by the absorption of the substances referred to varies within extraordinarily wide limits. Some persons are remarkably sensitive to the toxins under consideration. Old people are more sensitive than young or middle-aged people. It might naturally be assumed that individuals of the definitely nervous type would always suffer most, and that the labouring class would be but little susceptible to them. While this assumption cannot be altogether set aside as incorrect, I am often surprised to find amongst the out-patients coming to me at the Royal Infirmary individuals of the labouring class suffering so profoundly from intestinal auto-intoxication as to be unfit for work. In some persons the manifestations of intoxication are so pronounced that they indicate a definite idiosyncrasy, an individual chemico-vital susceptibility, which is not, so far as can be seen, a matter determined by social class distinctions or by occupation.

The question that emerges from the relations here submitted as subsisting between alimentation, the fate of food

in the alimentary tract, and the effect upon the composition of the blood of absorption from that tract is: How much is the effect upon the vessels to be attributed to the normal reflex acting through the vasomotor centre in the medulla, and how much to the direct influence on the vessels of substances present in the blood? Which, for instance, of these is the main and leading factor in constipation? Does the retention of excrement in the intestine continue so to stimulate the splanchnic system that the stimulation of the centre for vasomotor constriction is continuously maintained; or, on the other hand, does the continuous absorption into and therefore the presence of noxious substances in the blood bring these substances into direct contact with the vessel wall, stimulating or exciting it to a hypertonic contraction? It is probable that both factors operate. There is no doubt as to the nervous reflex, and there is little doubt as to the other factor. It has been already shown that certain substances added to the blood lead to vessel contraction without the aid of the nervous system. It is of course always interesting to know the precise steps by which effects which interest us are obtained; but it remains to be settled whether the substances to which we refer and which cause vessel constriction act through the nervous apparatus or more immediately upon the muscle tissue of the vessel wall. Even in this connection it is, however, to be borne in mind that when there exists a complex nervous mechanism it becomes involved in processes, although a like result can be obtained without its participation. This is shown by the fact referred to earlier that the tone of arteries is soon restored after section of vasomotor nerves. I do not claim that the substances which act as it is here held they do, have been individually and severally separated and investigated; and yet I do not think any physician of experience questions the correctness of the general belief in "blood impurity," and that it commonly has its rise in disorder of the alimentary system, or is brought about, directly or indirectly, by absorption from that tract. The muddy complexion, the "dirty" conjunctiva, the face pimples, are the visible manifestations of the accuracy of the popular conception. That the blood-content is influenced by what is taken into the alimentary

system has much support from the common knowledge as to the production of urticaria by the ingestion of certain substances by certain persons. The extreme subtlety of blood-content is realised when a little shell-fish in one person, a few gooseberries or strawberries in another, will lead to an extensive urticaria which is only to be explained by the action of *something* brought to the surface by the blood. Although the *something* has not been separated and christened, it would be the merest scientific priggery to question its existence or to deny the sequence of phenomena. That deleterious substances enter the blood from the intestinal tract cannot be seriously questioned, and believing this, it is not necessary to occupy space in further argument in support of its truth. The acceptance of this carries the acceptance of the minor truth, that the amount or quantity of such deleterious substances varies within very wide limits, and that whatever effect they may have will equally vary in degree.

The fact on which I seek to lay great emphasis is that substances absorbed from the alimentary tract lead to vessel contraction. That fact seems to me to be fundamental and not open to question. To my mind it seems unreasonable to suppose that such substances cause vessel constriction only through the systemic vasomotor centre. It has been seen how erroneous is the common tendency to attribute all such vascular changes to that mechanism, and that experimental investigation has proved that even substances which belong to our most important medicinal remedies act not through that mechanism but directly upon the vessel wall. I contend further that clinical indications point to this direct influence on the vessel wall of substances absorbed from the intestinal tract, and that the common effect is a hypertonic contraction of the arteries. It is this arterial contraction which has not been recognised as the essential factor in the production of the "high tension" associated with certain conditions in the alimentary system, and which has been so much written about. I have failed to find any indication that the steps by which the "high tension" was brought about ever got beyond the nebulous idea of "peripheral resistance."

My contention at this stage may be thus stated: as a

result of improper or excessive feeding, of faulty digestion, or of defective bowel evacuation, substances are absorbed into the circulation which act upon the arterial and capillary walls, leading to an abnormal degree of contraction of the vessels. The effect varies with the intensity of the cause; the duration of the effect upon the continuance of the cause.

When the major proposition is grasped, it unerringly follows that there must be minor degrees of the condition we have pictured.

The acceptance of this proposition leads to the understanding of the beginning of things—it shows the first steps in a process which has only been known in its fully developed stage and for the explanation of which there has been a continuously expressed desire. The arterial contraction caused by substances present in the circulation is the first step in the causation of arterio-sclerosis; and, if there be no kidney disease, the substances which cause the contraction are absorbed from the alimentary tract, and are the product in one form or another of what has been swallowed in the form of necessary aliment or of unnecessary indulgence. It is necessary to state the position thus, for the question, as will presently be seen, is not confined to the simple point of excess in quantity or indulgence in what may be regarded as essentially injurious in virtue of its nature.

Behind the questions of quantity and character lie the factors of inefficiency or disorder of the gastro-intestinal functions. These are factors which it seems to me have been almost wholly overlooked. That indigestion in its various forms, particularly when associated, as it commonly is, with definite constipation, or with incomplete bowel evacuation, should be the commonest of all causes of vessel constriction gives to the disorder a significance which has not commonly been appreciated. In the great majority of cases of the kind indicated it will be found that arterial hypertonus is present. The thickening is not necessarily great, and, when it is associated with a somewhat small and feeble pulse wave, there is such a marked absence of what is known as *tension*, that the thickening is not even recognised, and so no deviation from the normal is noted, beyond the smallness of the pulse. In other cases, if the

thickening of the artery is recognised it is regarded as arterio-sclerosis, and when it occurs in a young or middle-aged person it is looked upon as an indication that so-called "senile degeneration" has already begun. Such a conception is undeniably prevalent, and gives support to its own falseness by ultimately leading to the result suggested. It becomes thus a matter of fundamental importance to recognise the hypertonic thickening of arteries; and to understand that the hypertonus, in such cases as are here indicated, is due to deleterious substances present in the blood irritating or stimulating the vessel wall; and that these substances are absorbed from the intestinal tract. The nature of these substances has been already referred to. The responsiveness of the arteries to them becomes very striking once the correctness of the proposition is tested.

This, I contend, is the first step towards the production of arterio-sclerosis in a vast number of instances. Whatever alimentary and intestinal conditions cause hypertonus only require to be continued to produce sclerosis, if cardiac vigour be at the same time sustained, as it frequently is.

It is perhaps necessary to definitely say that, while stating the foregoing proposition, it is not to be inferred that I mean anything beyond what I state. The spare feeder may develop arterio-sclerosis earlier than the big feeder, the moderate drinker develop greater vessel changes than the man who occasionally gets drunk. If the big feeder and moderate drinker has good digestion and free bowel evacuation daily, as such people frequently have, he may not devolop arterio-sclerosis till late in life. Nothing impedes advance so much as strong one-sided statements, which usually enjoy the position of maintaining but a fragment of truth. While arterio-sclerosis might be, on a partial view of the position, represented as merely a question of food and drink, the contention would be so misleading as to make it untrue, for it would leave out of account the spare feeder and the teetotaler, who, if he be a dyspeptic or the victim of habitual constipation, will assuredly supply in middle life a typical example of "premature arterial degeneration." It is the faculty of dealing efficiently with the intake, and the easy and complete removal of waste and

excrement, which postpone vessel changes in many a man who indulges his palate as his abstemious contemporary dare not do, under the risk of acute poisoning. It is here that the explanation lies of the marked diversity of opinion held as to the influence of food and of alcoholic drinks in producing vessel changes. It is a fact that neither the big flesh-eater, nor the man who daily partakes of wine, spirits, or beer, necessarily shows vessel changes early in life, and yet there seems to me to be no doubt that in other men these things lead to vessel changes. In the former class digestion is faultless and elimination perfect; in the latter, the first may hold and the second fail. So it is with the spare feeder: he may digest and eliminate efficiently up to his particular level and capacity; while an equally spare feeder may be using a dietary not suitable to his standard of digestive capacity and excretory efficiency: of the two, the former shows soft vessels, the latter thickened vessels.

When it is understood that it is not necessarily the character of the materials which determine the vessel changes, what has hitherto been a difficulty disappears. We need no longer consider which is the right and which the wrong view, for, as is usually found when the experience of competent observers is conflicting, they have each got only a bit of the truth. It is not any special poison in the material, but the presence of waste or of absorbed poison in the blood, that determines vessel changes—the waste may be the waste of the physiological wear of tissues; it may be the waste of unutilised pabulum, unused because excessive in amount. The name of the special purin body in the special food of the particular individual may be safely left to the pure chemical pathologist. For clinical and practical purposes the essential fact is that the tightening up of the arteries is due to the presence of substances in the blood which ought not to be there in any sufficient amount to keep up vessel constriction.

I even carry my contentions further than this. To me the influence of the condition of the blood upon the tone of the arteries is so certain that I have realised for some time that in many cases the recognition of arterial hypertonus suggests either disordered metabolism or constipation, and I

am struck with the number of instances in which the surr proves to be correct and becomes the basis of succes treatment.

There is thus produced a chain of graduated links, e link an example of the same underlying physiological prop tion, which must, I think, rank as a law—namely, that ve tone is influenced and determined by blood composition, that to us as practitioners of healing the important poir that blood composition is determined, directly or indirectly absorption from the intestinal tract.

It will not be altogether unprofitable to devote s further words to the consideration of blood composit When we do so we are forced to recognise that it must s much more subtle variations, as regards the extraneous stances which may be present in it, than the ordinary chem composition as supplied by physiology suggests. The bl content is in the first place influenced by the food ta The processes in digestion cannot be regarded as pic out and selecting merely what is suitable from wha swallowed: all substances are acted upon so that their sol constituents can be absorbed, either into the lymph syste directly into the blood system. In the latter case, the l serves not only as a living filter-bed but as a chem "clearing-house" for the blood brought by the portal from the alimentary tract, before it passes on into systemic system. When, on the other hand, substa pass directly into the lymph system, only the lymph gl interpose between them and the systemic blood syst and it is not known that these glands have any *chemical* to perform in this connection, although they filter out particles whether these be living or dead. In either there is of course no doubt that substances reach the b by one or other channel, which are not necessary for physiological requirements of the body. That this occu of course fully established by our everyday clinical obse tions and therapeutic procedures. The fact that certain f in some persons produce urticaria is abundant proof of even were there no other. The influence of alcohol, effects produced by the drugs we use, are all brought a by the passage into the blood of the unmodified substa

themselves, or as they are modified in their passage through the alimentary system; for it is to be realised that both processes occur.

Although this is so evident that I do not suppose it will be questioned in any quarter, I am not aware that physiological chemistry is in a position to determine what the subtle substances are in the blood which produce urticaria after the ingestion of certain things. Yet it would be the veriest folly for the clinician to doubt the existence of such substances; and the clinician's observations ought to stimulate physiological and chemical research.

I say so much because I am not competent to discuss with any semblance of authority the subtle changes which must be acknowledged as occurring in the blood as the result of the various foods and fluids partaken of, or of the manner in which they are dealt with in the alimentary tract from the stomach onwards, including the final ejection of their refuse and waste. At the same time, I fully recognise the value of the investigations which have been made of the purin group of bodies by Dr. Walker Hall and others; and of the suggestiveness of the proposition that these bodies are contained in common articles of diet, pass from the alimentary tract practically unchanged into the circulation, and exercise certain undesirable influences. This is the direction in which research will no doubt be continued until knowledge becomes fuller and more assured; meanwhile, that work may already be accepted as confirmation of the major proposition that substances are taken up from the alimentary tract into the blood which directly influence the arterial and capillary walls. I have already dealt with the evidence from experimental investigation proving the direct effect of substances in the blood, or circulating fluid, upon the vessel wall, leading to increased tone or contraction; and that the common conception of the vessels merely responding to vasomotor nerve impulses has to be abandoned, and the wider proposition adopted. Having thus far carried my contentions, it remains to be shown what clinical evidence is available, not only in support of these contentions, but as indicating more definitely the dietetic substances which act most potently; whether they act in virtue of their essential composition, their

excessive quantity, or as a result of secondary changes taking place in them in the alimentary tract.

In the first place stands the question as to whether the kind of food is of any practical importance? There is no doubt that the great bulk of clinical opinion and experience, both in the past and the present, points to the more highly proteid foods producing effects not produced by the carbohydrate foods. Free and abundant flesh-eating has come to be regarded as leading to "heightening of blood-pressure" and to consequent arterial changes. This appears to be the view expressed in recent writings on subjects where blood-pressure and "arterial tension" are included in the consideration of the questions which are discussed. It is so prevalent that it would be invidious to single out individual examples when our aim is to deal with the whole question of blood-pressure and vessel-changes from the constructive side, and to avoid the polemical tone which destructive criticism can hardly avoid. With the common view as to the injurious effects of a large proteid dietary I wholly agree. At the same time, the common view requires important modifications to give it its full clinical value. The mental picture is too much confined to the man who eats largely and becomes florid and "plethoric" as a result—the man of Herculean digestion. For clinical purposes, it is essential to recognise the large class of persons who eat moderately, or are even regarded as small eaters, but whose digestive functions or alimentary-tract processes are faulty. In these persons even their spare proteid dietary is excessive. In some cases the error appears to be digestive, but more commonly the essential error is constipation. Even when there is digestive disorder, it may be the result of constipation as well as the cause of it. Proteid food retained in the intestine putrefies and raises the virulence of the colon group of bacilli, and the products are absorbed, as has been already indicated. In fact, the factor of putrefaction seems to me to be so common, as a result of the prevailing constipation, that I give it a foremost place. I do not find indications of proteid food being injurious in moderate feeders who have very free daily bowel evacuation. The cult that holds that no man eats flesh with impunity is as unscientific as extremes usually are. If the spare feeder,

in spite of his abstemiousness, suffers auto-intoxication from putrefying proteid, he will find material comfort in a dietary which does not so putrefy; but the primary fault is in the alimentary tract, not in the food itself. Putting aside the question of mere vulgar excess, what one finds in the class of case I have been dealing with is, that there are usually subjective symptoms of various kinds commonly attributed to "biliousness" or to "liver," that along with these the radial artery is hypertonic, and the urine indicates the presence of intestinal putrefaction. In such cases there is no doubt there are deleterious substances present in the blood, for they are excreted in the urine; and it seems to me certain that they act directly upon the arterial wall, leading to a hypertonic contraction of it. Certain it is that if the bowel be unloaded the hypertonus relaxes. A dose of mercury may have the same effect, even before it acts as a laxative, presumably owing to some influence it has directly or indirectly on the intestinal contents.

There is some difficulty in turning people's minds round to look not so much at absolute quantity as at the use made of what is taken. The importance of intestinal putrefaction has not been known; and it has not been widely realised that carbohydrate foods ferment but do not putrefy.

I have hitherto in vain sought for evidence that carbohydrate food leads to the arterial hypertonus which I have referred to as resulting from the putrefaction of proteid.

The position may be summarised thus: an excessive flesh dietary, even when digested, is hurtful because of the excess of material absorbed, which cannot be utilised and has to be eliminated. When putrefaction occurs, whether it be in well-digested or ill-digested flesh foods, the products are highly injurious. The injurious effect is shown in the arteries by hypertonic contraction and so-called "high tension," and this ultimately leads to arterio-sclerosis. Carbohydrates do not have the same kind of effect.

The symptoms which are popularly and vaguely defined as *gouty* or *suppressed gout* are all referable to the defects which have been indicated. They are all due to the presence of waste retained, or deleterious substances absorbed and not readily excreted. The mental depression, the physical

weariness, the bilious attack, all have their source here, and they will be found to be always accompanied by hypertonic arterial contraction. As we relax the vessels by treatment the symptoms disappear, and, if we can so order life as to prevent hypertonic contraction, there is an absence of the *gouty* symptoms.

Believing the position to be such as is here indicated, it follows that I place the condition of the alimentary tract in the forefront of the causes which lead to arterio-sclerosis, with all its manifestations, and all its disastrous consequences, including the shortening of life.

I cannot pass from the alimentary tract without referring specifically to the effect of beer and spirit drinking upon the vessels. Acute alcoholism is dealt with in a separate chapter, but from what I have seen there seems to me to be no doubt that in some persons the steady daily use of beer or whisky leads to pronounced arterio-sclerosis. I append a short note of a few cases illustrating this point. It is not the invariable result in all persons, as will be seen from the subsequent chapter dealing with alcoholism. The fact is that the effects of alcohol afford but another illustration of what I have already tried to enforce, that it is altogether wrong to take up the position that the same substance produces the same physiological or pathological results in all persons. An excessive proteid dietary is entitled to rank alongside alcohol; but it is probable that constipation is the cause of as much arterial change in the people of Britain as either the one or the other.

THE EFFECT OF THE ALIMENTARY SYSTEM ON SCLEROSED VESSELS

From what has been said with regard to the influence of the alimentary system in leading to hypertonus and ultimately to sclerosis, and bearing in mind the contention that sclerosed vessels readily become hypertonic, the further statement will be anticipated, namely, that when sclerosis is established, the same circumstances or conditions in the alimentary tract continue to act. The point is, however, of such great practical importance that it is desirable to accentuate it by specially

referring to it here. In elderly people with sclerosed vessels the influence of this tract is very striking, when the recognition of hypertonus becomes a matter of routine observation. The effect of proteid food in them is in my experience quite beyond question. Whether, however, it be the supply of proteid in excess of the needs of the inactive body, or intestinal putrefaction and constipation which supplies the precise irritant, is not easy to determine; but in many cases the two factors are conjoined.

The sclerosed vessels are indeed remarkably sensitive to the irritant or stimulant, whatever it be. I do not attempt to offer any explanation, but I am convinced that it is a clinical fact. The sensitiveness may become marked even to impressions conveyed through the vasomotor centre, as I have shown in Chapter XV., when dealing with angina pectoris. The practical and clinical bearing of the fact is that the irritability of the vessels can be in a great measure controlled by an austere dietary, conducted on the lines indicated by recent and more accurate physiological investigations.

In elderly or old people it will be found that there is no surer guide to the right line to be taken in their management and treatment than by the recognition of whether hypertonus is or is not present.

In subsequent chapters cases are given in which the influence of food or of constipation is shown; and on the other hand, the effect of bowel evacuation is seen. It seems, therefore, unnecessary to elaborate this matter more fully here, but I hope I have said enough to carry the conviction of the importance of this side of our subject. As having some bearing upon the following section, I may say that I have tried thyroid in these cases, and it seemed so definitely unsatisfactory that I have ceased its use.

THE INFLUENCE OF THE ALIMENTARY SYSTEM ON THE ADRENALS

In an address delivered to the Derby Medical Society, and published in the *British Medical Journal* on 4th June 1904, I suggested that the effect of a rich proteid diet in causing hypertonic contraction of vessels might act by stimulating

the secretion of the suprarenal glands, extracts of their medullary portion being known to have this constrictor action on the vessels. Three years of added experience have drawn me further away from this idea; while the effect of absorption directly into the blood from the intestinal tract seems a more satisfactory explanation of such a clinical phenomenon as the removal of hypertonus by laxatives. As these pages, however, were being corrected for the press, the very able and suggestive address delivered by Dr. H. D. Rolleston to the Canadian Medical Association appeared in *The Lancet*. In that address he referred to the suggestion mentioned above. It is not, however, for that reason I refer to it now, but because his address, while presenting a vivid picture of experimental and clinical observations on the problems which surround the suprarenals, brought back an idea which suggested itself to me years ago, when, as pathologist to the Royal Infirmary, I was methodically examining these glands. The idea was that the cortical part of the gland was for the destruction or transformation of the substances present in the medullary portion. This idea would explain the apparently curious fact that extracts of the latter have such a marked physiological action, while, so far as is known, the cortex has no such action. Following this idea further, it may be that the active principle in the medulla may be taken from the blood as a harmful substance, and altered in its passage through the cells of the cortex into a harmless substance. Were this found to be the true view, it would easily follow that an excess of proteid absorption or of other substances from the intestine throws a greater strain on the glands than they were equal to, and that constrictor substances accumulate by retention in the blood. As giving some little support to this suggestion, I may add my impression that in advanced life the cortical part of these glands is relatively small. If these speculations turned out to be correct, they would explain the sensitiveness of sclerosed vessels in elderly persons referred to in the previous section. In this particular region of speculation no harm follows a wrong view, and, when Dr. Rolleston regards his imaginings as "somewhat wild speculation," I would remark, that such speculations may become the facts of a later date. It would only mean that

the glands were excreting and not producing the pressor substance found in their medullary part.

CASES ILLUSTRATING THE FOREGOING CONTENTIONS

Excessive proteid dietary with thick and tortuous radials—recurring aphasia

CASE 10.—R. S. K., a man in middle life, sent by Dr. M'Ewan of Prestonpans, with a history of temporary attacks of partial and complete aphasia. The radial arteries were very thick and tortuous. The heart extended to an inch beyond the nipple line. The urine contained no albumin, the specific gravity was 1011. He usually had to get up in the night to pass water. On inquiry, he acknowledged that he had "lived on beef"; he took beefsteak to breakfast, broth and beef to dinner, and beef to supper. There was no other factor in his history to explain the arterial thickening.

Excessive proteid dietary with thick radial arteries in a young man

CASE 11.—T. N., æt. 25, butcher, Fife, seen at the Royal Infirmary, 15th November 1905. He complained of pain in the stomach in the forenoon, and sometimes in the evening. The pain at first was in the pit of the stomach, but more recently it was "working to the left." The bowels moved daily. The doctor he had consulted thought the pain was due to a strain. The radial arteries were markedly thickened. The heart was normal, and there was no albumin in the urine. The thickening of his vessels was so marked, and as there was no evidence or history of syphilis, I made special inquiries as to his diet, and found it to be as follows: at 7 a.m. he had bread and butter and tea; "breakfast" was at 12 noon, and consisted of half a pound of steak and an egg; "dinner," at 2.30 p.m., consisted of soup, another half-pound of meat, potatoes, a glass of milk, and rice; "supper," 7 to 8 p.m., consisted of fish, bread, and tea.—I impressed upon him the fact that he was doing himself injury by the amount of flesh he was taking. I advised him to take meat once a day only, and prescribed 5 grains of potassium iodide thrice daily.

Constipation with auto-intoxication and hypertonic arteries

CASE 12.—Wm. A., æt. 41, an iron moulder, sent to the Royal Infirmary from Falkirk, complained of weariness and want of strength, and as having had a cold with a little cough for some months. He brought a specimen of his sputum, but it was quite colourless, and we did not consider it necessary to stain it for tubercle bacilli. Examination of his lungs and heart showed them to be apparently healthy. The tongue was clean, with moist edge and dryish centre. He had indefinite sensations in the abdomen at times, but he had evidently paid no special attention to them, and could neither locate them nor describe them. He had much flatulence. The stomach was dilated, reaching as low as the umbilicus, and although it was four hours since he breakfasted, his stomach was full and splashing. The urine contained no albumin or sugar. The bowels moved nearly daily, but he had to regulate them by taking laxatives. The radial arteries were thickened and definitely hypertonic. —My opinion of this case was that the patient had atonic dyspepsia with gastric dilatation; a measure of constipation; that the languor and sense of weakness were the result of auto-intoxication, and that the hypertonic condition of the arteries was to be similarly explained.

Hypertonus from constipation

CASE 13.—Mr. T., æt. 30, was being examined for insurance. He did not smoke; there was no sugar or albumin in the urine; the heart was normal. He had quite definite hypertonus of the radial artery, and this was associated with a distinctly costive habit.

Constipation with precordial pain and hypertonic radials

CASE 14.—N. P., iron moulder, æt. 20. This youth was admitted to hospital suffering from recurring pain and discomfort in the precordia along with symptoms of gastric disorder. He was constipated, and there was marked hypertonic thickening of his radials. The bowels were regulated by a daily dose of cascara and glycerine, and a

rhubarb and soda mixture were given for the stomach condition. The pain disappeared, and the thickening of his arteries almost entirely.

Cases illustrating the effect of beer-drinking on the vessels

CASE 15.—Henry F., aged 36, engineer, seen at the Royal Infirmary on 5th September 1906. He was a repairer of bookbinding machines, and therefore travelled a good deal. He complained of pain in the back when he stooped, and lately of great giddiness, especially when stooping. The giddiness had been so severe that he volunteered the statement that he had nearly fallen down the hoist track. He was a vigorous, intelligent-looking man, with a sallow complexion, and icteric-tinged conjunctiva. He was married, and had six healthy children. The bowels moved daily, but he "suffers terribly" from flatulence passing by the bowel. The tongue was thickly coated in its posterior two-thirds. There was nothing to note in the thorax or abdomen. The urine contained neither albumin nor sugar: he said it varied in colour: the sample we saw was rather pale in colour. The radial arteries were markedly thickened and hard. He was an Englishman, and every night nearly drank two or three glasses of beer. He never exceeded. He had no appetite for breakfast.—I prescribed for him a mixture containing salicylate of sodium and iodide of potassium to be taken thrice daily; a laxative pill to be taken nightly; and strongly advised him to stop his beer.

CASE 16.—Thos. N., aged 37, seen at the Royal Infirmary on 5th September 1906, brewery carter, delivering casks of beer to the public-houses in the city. Complained of heaviness in his belly and of being "awful giddy whiles." He is a big, stout, heavy man. There was a little tremulousness about the mouth when he was being questioned about his beer-drinking. There was nothing to be made out in the abdomen. The bowels moved daily. He was not sure as to the amount of beer he drank daily, but it seemed certain that his habit was to drink whenever he had the opportunity, either at the brewery or when offered it at the public-houses at which he

delivered. He had been over twenty years working about breweries. The urine contained no albumin. The radial arteries were so thick and hard that they might almost have been called rigid—they were comparatively small vessels, as if they were diminished in size from hypertonic contraction.—I ordered him a laxative pill to be taken nightly for a week; 20 minims of sp. etheris nitrosi to be taken thrice daily, to give up beer absolutely, and to report himself to me in a few days.

10*th September.*—Reported himself at the ward. Feeling much better. Giddiness gone. Radial much larger and more compressible—now a thick vessel not suggesting rigidity: Oliver pressure, 170; Gärtner, 150.

Extreme arterio-sclerosis from the daily use of alcohol for many years

CASE. 17.—Wm S., æt. 64, came to the Infirmary complaining of pain in the back of his head, in the temples, and over the vertex, also of giddiness. He was a healthy-looking, well-nourished man with a slightly florid complexion, but older-looking than his years. Both temporal arteries were prominent and were the thickest and hardest temporals I had ever seen—in size and hardness they almost felt like pen-holders. The radial arteries were in a similar condition, although not quite so extreme. There was no albumin in the urine. He assured me he had always been a steady man, had never drunk to excess or to do himself "any harm." There was no history of syphilis, of rheumatism, or of gout. He never suffered from dyspepsia, and his bowels always moved daily and freely. In fact, there seemed to be no explanation obtainable of the extreme sclerosis. With more detailed questioning it came out that up until within the last few years he had spent his working years since a lad as an assistant to licensed grocers and wine merchants, and that during all those years he had daily taken spirits.

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CHAPTER XIII

HÆMOMANOMETER PRESSURES IN CASES OF ACUTE ALCOHOLISM

THE VALUE OF A VASO-DILATOR ADDED TO THE ORDINARY TREATMENT.

In this chapter I propose to record observations made by myself and my clinical assistant, Dr. Green, on patients in Ward 3 of the Edinburgh Royal Infirmary, and to consider these observations as illustrating and supporting my main contentions regarding the records of so-called blood-pressure. At the same time it will be shown that here also the records are of much interest, and, when rightly understood, of decided clinical value.

The cases to be dealt with were cases of acute alcoholism, differing in intensity, but all sufficiently pronounced to have been admitted to the ward provided for the reception of patients suffering from "incidental delirium." The patients whose conditions are dealt with were all in the midst of a bout of heavy drinking, either the victims of hallucinations of sight or hearing, or in the stage of yattering incoherence with tremor and restlessness. In the stage of tremor and constant muscular movement it was of course impossible to make accurate hæmomanometer observations; but this phase might not develop until the patient had been in hospital for twenty-four hours or longer: in one case it did not develop until he had been in the ward for three days; and, having been in on several previous occasions, it was known that this was the usual time for his delirium to begin. In this way hæmomanometer readings were sometimes obtained before delirium developed.

The first generalisation I have made from these cases is

that not in all cases is the arterial pressure high; the second is that in the great majority of cases the pressure is relatively high to begin with, and falls as the patient improves. In the latter group the pressure was usually about 150 and fell to 115 to 125 or thereby, if the arteries were not sclerosed. When they were sclerosed the readings to begin with were much higher than this, and did not fall as low as 150 although the patient had recovered.

GROUP I.—CASES WITH LOW PRESSURES.

As examples of what may be regarded as low pressure in acute alcoholism, the following cases may be given:—

CASE 18.—A. A., male, aged 36, was seen by one of the resident physicians, and admitted to Ward 3 on 8th January 1907. On admission the patient was excited, with a rapid pulse, a moist skin, and was stated to have spoken of seeing various animals crawling on the walls. He was given 30 grains of bromide of potassium and 20 grains of chloral hydrate. He was admitted at 9.45 p.m., but he was not asleep until 4 a.m. When I saw him on the 9th his pressure at 12.30 p.m. was only 115, the radial artery was soft, and the pulse of fair strength. He was still very tremulous. He was ordered a mixture containing strychnine, digitalis, and valerian. On the 13th his pressure was 95; he was feeling very well. He was allowed to go home on the 14th.

CASE 19.—W.M., aged 33, maltman, was admitted to Ward 3 on 14th January 1907. He drank beer and whisky. On admission he was restless and tremulous, and was given 20 grains of chloral. On the 15th when I saw him his pressure was 120. He was a plump man with a plump, fat arm. On the 17th the pressure had fallen to 110, the vessel wall being soft with no thickening.

The following four cases of acute alcoholism further illustrate the state of the arterial wall on admission, the treatment given, and the change in pressure after treatment.

In all it will be noted that the pressure, although not high on admission, fell still lower on recovery. In all four cases the relaxation and softening of the arterial wall was appreciable to the finger as the pressure fell.

Sex and age.	Condition of radial artery.	Treatment.	Date.	Pressure.
Male 30	Tightened up	Stomach lavage	March 15	120 mm. Hg.
			,, 16	105
Do. 44	Do.	Erythrol and chloral	Jan. 23	135
			,, 29	105
Do. 38	Do.	Erythrol and trional	,, 18	115 (taken after treatment begun)
			,, 22	95
Do. 50	Do.	Iodide, squill, and digitalis	March 8	115
			,, 14	105
			,, 15	100

GROUP II.—CASES IN WHICH PRESSURE WAS RELATIVELY HIGH. THE EFFECT OF A VASO-DILATOR ADDED TO A HYPNOTIC.

The following cases belong to the more common type, in which the pressure was relatively high on admission and fell to normal or to some 10 degrees or so short of normal. Some of the cases illustrate the effect of a combination of erythrol tetranitrate with hypnotics—a combination which was suggested by my clinical assistant, who argued that the sedatives we were using might act more promptly if the vessels were at the same time more promptly dilated by means of erythrol tetranitrate. This combination has given us in some instances strikingly satisfactory results. We have so far only followed this method where the hæmomanometer readings have been high, but propose testing the effect of the combination on a more extensive scale.

CASE 20.—R. C., male, aged 46, was admitted to Ward 3 by a house physician on 7th January 1907 at 11.30 p.m., suffering from delirium tremens—very tremulous, and seeing and picking up imaginary coins from the coverlet of his bed. At 1 a.m. on the 8th he was given 20 grains of chloral, a like

dose being given at 5 a.m. He did not sleep but was quiet and muttering low. When I saw him about noon he was tremulous, in muttering delirium, and his arteries were like whipcord. At 1 p.m. he was given 20 grains of trional, half a grain of erythrol tetranitrate, and 10 minims of tincture of digitalis and tincture of squills: as he was not asleep, the erythrol, digitalis, and squill were repeated at 5 p.m.; at 6.45 he was asleep, and slept all night. On the 9th he was still very tremulous, but the mental condition had improved; his radials were decidedly softer, but the pressure was 150. He had 3 doses of the medicines last mentioned during the day. 10th January—slept all night; mental condition quite clear: arteries soft, pressure 120. The erythrol was stopped but the digitalis and squill continued. 12th January—the patient had completely recovered; the pressure was 115, the radial arteries relaxed and soft. On the day of admission, when the arteries were so tightened up that they felt like whipcord, the pressure cannot have been less than 180; but, owing to the patient's condition, it was quite impossible to take a reading.

CASE 21.—J. H., male, aged 48, was admitted by a house physician on 11th January 1907 to Ward 3, suffering from delirium tremens; he was tremulous, and had hallucinations of vision. The patient had been drinking heavily for some time. He was admitted at 5 a.m. I saw him in the forenoon, and found his pressure at 11.30 was 150, the pulse was 96, the radial artery large and well filled, with a thick wall and a large wave. He was given ¼ grain of erythrol with 10 minims each of tinct. digitalis and tinct. scillæ at noon; at 12.45 the pressure was 130. At 6.20 p.m. the pressure was 140, at 6.30 the erythrol, digitalis, and squill were repeated, and at 8 the pressure was 130. At 11 o'clock, as he was excited and restless, he was given 20 grains of trional; at 11.45 he was asleep, and slept until 5 o'clock the following morning. On 12th January, at 11.30 a.m. the pressure was 125; on 13th January, at 12.30 p.m. it was 125; on 15th January, at 1.20 p.m. it was 125; and on 17th January, at 12.30 p.m. it was 125. The relaxation and softening of the vessel wall which

accompanied the fall in the hæmomanometer readings were quite perceptible to the finger. In this patient, who in ordinary circumstances would have had a day or two of acute delirium tremens, the attack was aborted by a single 20-grain dose of trional given after his vessels were relaxed by erythrol.

CASE 22.—D. F., male, aged 49, was admitted on 14th January 1907 to Ward 3. This man came to the ward of his own accord, and sought admission as he knew he was to have an attack of delirium tremens. He had been in the ward on several previous occasions. He presented himself in the period of abnormal calm which in him was known to precede the alcoholic storm. It was not until the 17th that he showed any cerebral symptoms: he then began to hold conversations with imaginary people, and between then and the 18th he became wildly restless and delirious. Erythrol and trional combined had no effect upon him, but four 20-grain doses of chloral put him asleep between the 18th and the 19th; and by 12.30 p.m. on the 19th he was awake perfectly sensible, and remained so. On former occasions much more chloral had been used, and the duration of the delirium had been longer. The pressure on the 15th was 155; on the 17th, 140; on the 19th, 140; and this notwithstanding several ½-grain doses of erythrol. On the 29th it had fallen to 125, the pulse was 68, the vessel soft and compressible. On the 15th, the day after admission, the frequency of the pulse varied from 85 to 105, and the vessel wall was thick and contracted. The influence of the erythrol was very striking in this case, for on no previous occasion had his delirium been so cut short, and convalescence been so rapid. Chloral acted better than trional with this patient. The reduction of pressure from 140 mm. Hg. to 125 was effected by means of iodide of potassium, and corresponded with the complete relaxation of the radial artery. This, indeed, was a case in which I thought there must be permanent thickening, and the very definite effect of the iodide was a surprise to myself.

CASE 23.—G. H., male, aged 31, admitted on 15th March

1907 to Ward 3, having drunk heavily and having taken a "fit." On admission he was tremulous and nervous—the radial artery was small and hard, the pressure was 150. At 11.45 a.m. he was given erythrol gr. ½ and trional gr. xx, but as he vomited ten minutes after they were taken, the dose was repeated at 3 p.m. At 4.15 p.m. he was asleep, and slept almost continuously till 5.30 the following morning. In the forenoon the pressure was 130, the radials were relaxed, and the pulse wave larger. This patient was practically well, and went out on the 18th.

These cases when arranged as below show how naturally they fall into two groups; while the fall in pressure in each member of the two groups is seen at a glance.

GROUP I.—*Low Pressures.*

Male, aged	36.	115	mm. Hg.	fell to	95	mm. Hg.	= fall of	20	mm.	Hg.
,, ,,	33.	120	,,	,,	110	,,	,,	10	,,	
,, ,,	30.	120	,,	,,	105	,,	,,	15	,,	
,, ,,	44.	135	,,	,,	105	,,	,,	30	,,	
,, ,,	38.	115	,,	,,	95	,,	,,	20	,,	
,, ,,	50.	115	,,	,,	100	,,	,,	15	,,	

Average fall, 18·3 mm. Hg.

GROUP II.—*High Pressures.*

Male, aged	46.	150	mm. Hg.	fell to	115	mm. Hg.	= fall of	35	mm.	Hg.
,, ,,	48.	150	,,	,,	125	,,	,,	25	,,	
,, ,,	49.	155	,,	,,	125	,,	,,	30	,,	
,, ,,	31.	150	,,	,,	130	,,	,,	20	,,	

Average fall, 27·5 mm. Hg.

CHAPTER XIV

GROUPS OF CASES ILLUSTRATING HÆMO-MANOMETER READINGS

1. FOUR CONVALESCENTS FROM ACUTE BRIGHT;
2. SEVEN MISCELLANEOUS CASES;
3. THIRTEEN ELDERLY AND AGED PERSONS: THE RELATION BETWEEN THE RADIAL AND BRACHIAL ARTERIES;
4. EIGHT CASES WITH HEART SYMPTOMS: THE IMPORTANCE OF RECOGNISING HYPERTONUS IN CARDIAC CASES.

CONVALESCENTS FROM ACUTE NEPHRITIS.

THIS series of cases illustrative of hæmomanometer readings may be begun with the following 4 cases, in which there was a definite diagnosis of acute Bright's disease, the patients having been admitted into hospital with œdema, and albumin, blood, and casts in the urine.

Case.	Age and Sex.	Disease.	Condition of Vessel Wall.	Pressure.	
				Oliver.	Gärtner.
				Mm. Hg.	Mm. Hg.
CASE 24	Male, aged 62	Acute Bright; convalescing	Thickened and sclerosed	165	150
,, 25	,, ,, 44	Do. improved	Do.	150	130
,, 26	,, ,, 40	Do. do.	Slight thickening	130	100
,, 27	,, ,, 9	Do. convalescing	No sclerosis	110	...

Of these the two first had thickened and sclerosed vessels, but not nearly so thick as were the vessels in the series of

kidney cases in Chapter XI.; in the third, the arteries were but slightly thickened; while in the fourth, a boy of 9 years of age, there was no sclerosis. The state of the arterial wall corresponded therefore with the manometer readings. It will also be observed that from the age of the individuals no definite inference can be drawn, for while there was a pressure difference of 15 between the ages of 62 and 44, there was a difference of 20 between 40 and 44, and a like difference of 20 between the ages of 9 and 40. The factor of age will be dealt with more fully later.

SEVEN PATIENTS UNDER OBSERVATION AT THE SAME TIME: PRESSURE CORRESPONDING WITH THICKNESS OF ARTERIAL WALL.

The following list comprises seven patients who were under observation at the same time as hospital patients:—

Case.	Age and Sex.	Disease.	Condition of Vessel Wall.	Pressure.	
				Oliver.	Gärtner.
				Mm. Hg.	Mm. Hg.
CASE 28	Male, aged 37	Brewery worker for 20 years. Vertigo; no alb.	Thick hard vessel	170	150
,, 29	,, ,, 50	Cabman, convalescent from jaundice; no alb.	Thick vessel	165	159
,, 30	,, ,, 69	Hydrothorax, etc.; alb.	Slight vessel thickening	135	113
,, 31	,, ,, 64	Aortic and mitral systolic murmurs	Vessel a little thick; compressible	135	...
,, 32	,, ,, 55	Anæmia, etc.; no alb.	Slight vessel thickening	125	80
,, 33	,, ,, 40	Sciatica, been resting for 6 weeks; no alb.	Soft vessel	110	90
,, 34	,, ,, 26	Muscular rheumatism; alb.	Soft vessel	105	70

These cases illustrate the general rule—namely, that if

you take the patients in a hospital ward, the manometer readings will be found to closely correspond with the state of the radial wall. Any other group of persons will give a corresponding result.

In Case 28, the brewery worker, observations were made after he had been for a week on spirit of nitrous ether and a nightly laxative. His vertigo under this treatment was cured, and he was feeling well. The radial artery was not quite so hard as it had been, but it was still much thickened, and a further week of treatment did not reduce the manometer reading. In fact, this man had at the age of 37 permanently thickened arteries, due, no doubt, to 20 years of steady beer-drinking.

In Case 29, a cabman, the observation given was made

A GROUP OF ELDERLY AND AGED PERSONS.

Case.	Age and Sex.	Oliver.	Condition of Radial.	Condition of Patient.
		Mm. Hg.		
Case 35	Mrs. C., Q.H., aged 68 . .	260	Thick and hard	Engaged in house work
,, 36	J. F., male, Q.H., aged 72 .	210	Thick artery	Has had hemiparesis
,, 37	R. C., male, Q.H., aged 72 .	235	Thick and hard	Supra-pubic cystotomy
,, 38	A.S., male, Q.H., aged 58 .	200	Thick	Old hemiparesis
,, 39	G. G., female, Q.H., aged 69 .	200	Hard, atheromatous and calcareous	Demented
,, 40	Mrs. R., Q.H., aged 92 . .	170	Soft and small vessel	Has all her faculties, and out of bed daily
,, 41	Mrs. B., Q.H., aged . .	155	Cordy and tortuous	Confined to bed
,, 42	Mrs. R., private, aged 80 .	160	Some thickening	Fairly active
,, 43	Mrs. S., private, aged 82 .	160	Thick	Do.
,, 44	Mrs. J., Q.H., aged 64 . .	140	Soft	Secondary anæmia from malignant disease
,, 45	Mrs. H., private, aged 70 .	130	Soft and compressible	Good health
,, 46	Mrs. I., private, aged 70 .	125	Do.	Fairly vigorous
,, 47	Mrs. M., private, aged 69 .	120	Do.	Do.

after he had had pilocarpine, and indicated the irreducible minimum below which no reduction of manometer reading would be obtained, because of the degree of permanent thickening in the vessels.

The age of the patients in this group also has no fixed relation to the manometer readings. The highest reading was from a man of 37, one of the lowest from a man of 40. In the two patients with readings of 110 and 105 the vessel wall was soft and unthickened. The intermediate cases with readings of 125 and 135 showed some thickening of the vessel, whether permanent or merely due to hypertonic contraction is immaterial so far as the conclusions drawn from the readings are concerned.

See Table on p. 121.

The cases on p. 121 show in the first place how erroneous is the idea that blood-pressure rises with age; and secondly, that if vessels are thick, hæmomanometer readings are high; if not thickened, they are normal.

The Relation between the Radial and Brachial Arteries.—Such readings could of course be multiplied almost indefinitely, but I do not think this would serve any useful purpose. That the readings represent anything like the variations in blood-pressure which the figures show, I can hardly imagine anyone with a cultivated knowledge of the pulse maintaining. The pressures corresponded, not with blood-pressure as perceived through the cultivated finger, but with the palpable measure of thickening or hardness of the radial artery at the wrist. There are, however, occasional cases where this proposition does not hold. Sometimes a patient with a thick radial gives a moderate degree of pressure, as in Case 41, where with a cordy radial the pressure was only 155: in some other cases, with a soft and compressible radial, the pressure is relatively high, as in Case 44, where with a soft pulse in advanced debility from malignant disease the pressure was 144. I have not found that the plumpness or leanness of the upper arm materially influences the readings. So far as I can see, the explanation is to be found by assuming that the condition of the wall of the brachial artery, which of course is the vessel compressed, does not correspond

with that of the radial as revealed by manipulation. Support is given to this as the probable explanation by the fact that occasionally one finds that the radial artery at the wrist may be distinctly softer than it is in the middle of the forearm. The general proposition that the pressure will be found to closely correspond with the condition of the radial wall, will however be found to be sound. So definitely is this the case that in many instances the pressure can be approximately guessed, after some experience, while at the same time it must be recognised that it is not wise to be overconfident. The position may be restated as follows: It is to the upper arm that the compression bag is applied, and when the pulse at the wrist stops, it is owing to the obliteration of the flow in the brachial artery. The wall of the brachial artery varies in thickness as the result of sclerosis, of atheroma, and of hypertonic contraction. The variations in its thickness determine the amount of pressure necessary to compress it so as to arrest the radial pulse. In the great majority of cases the amount of pressure necessary to obliterate the brachial can be judged of by the thickness of the radial artery; but, in a small number of cases, these do not correspond, and the only possible explanation is that the radial is not in them a reliable index of the condition of the brachial artery. The second case (Case 36) in the preceding list is an example of this. He had a moderately thick radial artery with moderate pressure in it, so that I thought it was hardly necessary to take his pressure. I did so, however, as I intended giving erythrol tetranitrate on account of the recurrence of a slight right hemiparesis, and it was much to my surprise to find that the obliterating pressure was as high as 240–250, and that it remained at that after days of erythrol. The erythrol seemed to distinctly improve his condition, although his brachial pressure remained the same. This man's brachial artery felt hard and difficult to compress by the fingers; but I state this with reserve, for I have not yet studied the brachial artery sufficiently to have confidence in the value of observations made by my fingers. The brachial is, as a matter of fact, much more difficult to gauge by the finger than the radial, as anyone can prove by trying

it. The following is a sphygmographic tracing from this man's radial artery:—

Since the above was written further developments have occurred which are worthy of note. On the 6th of September

FIG. 31.—Tracing from Case 36. Hæmomanometer Pressure, 200 mm. Hg.

the patient developed left hemiparesis, and I had him transferred to my ward in the Infirmary. On admission his pressure was found to be 240 mm. Hg.; under erythrol it fell to 220 mm. Hg. The condition of the radial artery did not suggest a pressure over 160 mm. Hg. On again carefully palpating the brachial artery it was felt to be very thick-walled and tortuous, this condition of the artery being both felt and seen at the bend of the elbow. It was as easily appreciated by others as by myself. I find that the best way of palpating the brachial artery is to pass the fingers round the back of the arm to its inner side, the observer using the left hand for the patient's right arm as he stands on the patient's right side. In the upper third of the arm the vessel can as a rule be readily felt and the condition of its wall estimated. In this case there was a difference of 30 mm. Hg. between the obliterating pressure in the upper and lower arm.

This want of relation between the radial pulse and the compressibility of the brachial artery as measured by the hæmomanometer has no doubt been the source of the proposition that the finger is an unreliable test of blood-pressure. It is a comfort to think that the use of the finger is not yet obsolete, and that the claim of the hæmomanometer to a more modest place may the more easily establish its true value as a clinical instrument.

EIGHT CASES WITH HEART SYMPTOMS.

In this series of cases the same proposition holds—namely,

that the hæmomanometer readings correspond closely with the condition of the arterial wall. The most striking example is in Case 54: the patient was an old man brought in a state of collapse to my ward in the Infirmary by the police. He rallied under judicious stimulation administered by the house physician. His arteries were thick and hooped, with anything but a vigorous wave inside them, and yet four days after admission his hæmomanometer pressure was 210. At the other extreme is a man of 63 with a soft compressible artery and a hæmomanometer pressure of 95 only. I doubt if there were 20 mm. Hg. between the *real* pressures in these two cases, and yet the hæmomanometer gave a difference of 115 mm. Hg.!

Case.	Sex and Age.	Condition of Radial Artery.	Oliver.
			Mm. Hg.
Case 48	Male, aged 64	Artery a little thick (angina pectoris)	135
,, 49	,, ,, 64	Artery thick (double mitral)	160
,, 50	,, ,, 57	Small—thready (double mitral)	120
,, 51	,, ,, 41	Artery thick (double aortic)	145
,, 52	,, ,, 59	Slight thickening (double aortic)	125
,, 53	,, ,, 46	Do. (mitral and aortic systolic)	125
,, 54	,, aged	Thick and hooped (4 days after collapse from heart failure, alb.)	210
,, 55	,, ,, 63	Soft compressible artery—feeble heart—splenic leukæmia	95

With regard to the other cases, five of which were under treatment for symptoms referable to valvular lesion, the condition of the artery is seen to correspond with the hæmomanometer readings.

The Importance of recognising Hypertonus in Heart-Cases.—It is desirable not to pass from this group of cases without drawing attention to the very great importance of recognising hypertonus in heart cases. There is nothing about it in literature, so far as I have seen. Sir James Barr has looked at the circulation from the periphery. Professor Saundby in a recent clinical lecture definitely refers to the value in some cases of heart disease of dilating the peripheral vessels. It is a fact which has been long known to me, and which I have used with much success. That the fact has not been more widely recognised is the result of the idea of

"peripheral obstruction" having been largely outside the "working principles" of daily practice. When it is properly recognised it will be often found that what a heart needs is not to be lashed by cardiac tonics, but to have measures taken for the relief of the hypertonic contraction of peripheral vessels, with its corresponding lowering of aortic blood-pressure. In heart as in other conditions the relaxation of hypertonus makes the arterial wall thinner, and there is a fall in hæmomanometer reading. Let me again warn those interested that relaxing treatment must not be adopted merely on the strength of hæmomanometer readings. A thick vessel with a corresponding low reading, say of 135, may require to have its tone raised; while a thinner-walled vessel, with say a reading of 125, may with advantage have its tone lowered slightly. The element of cultivated skill cannot be eliminated. The internal pressure, and the relation of the vessel wall to its contained blood, have to be judged of; and the daily hæmomanometer-reading merely helps the finger in the determination of the therapeutic course to be taken day by day.

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CHAPTER XV

THE RELATION OF ANGINA PECTORIS AND ALLIED CONDITIONS TO AN ARTERIO-CARDIAC REFLEX HAVING ITS ORIGIN IN THE ABDOMEN, AND CAUSING HYPERTONIC CONTRACTION [1]

INTRODUCTORY.—Angina pectoris has attracted the attention, and at times riveted the thought, of many clinicians during the past one hundred and thirty years. That it should still exercise the same influence over the clinicians of to-day need not, therefore, cause surprise. Gibson of Edinburgh, Oliver of Newcastle-upon-Tyne, and Mackenzie of Burnley have all three recently shown that they were the victims of the fascination or attractiveness of the subject. The views which are submitted here have emerged from the special attention which I have long given to cardiac and vascular disturbances, and more recently from a fuller knowledge of the processes of primary and secondary digestion, of nutrition, and of elimination. My indebtedness to the work of Pawlow, Herter, Chittenden, and others I gratefully acknowledge. Instead of conflicting with past observations, these views interpret them, and give the clue to the disentanglement of the confused mass of opinion on the subject.

Historical—*Heberden to Trousseau*, 1768–1862.—We do not require to go farther back in our historical retrospect than the date when the term "angina pectoris" was introduced into medical nomenclature. Those who wish to examine the earliest records of the symptoms called by this name will find the references in several of the older authors, particularly in Parry's monograph.

[1] This chapter is the reproduction of a paper, with some alterations, which was published in the *British Medical Journal*, 10th February 1906.

ANGINA PECTORIS

Angina pectoris was the name given by William Heberden, in the year 1768, to a disorder of the breast, which he thus described: "Those who are affected with it are seized while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away if it were to increase or to continue." "The seat of it and sense of strangling and anxiety with which it is attended may make it not improperly be called angina pectoris."

As regards the nature of the disease to which he gave this name, he says that "the opinion of its being a convulsion of the part affected will readily present itself to anyone" (p. 64).

In a later paper, of date 1785, containing the account of the *post-mortem* examination on the historical "unknown," he reports that there was no lesion found; and in his comments on treatment he advocated the use of medicines which "relieve and quiet convulsive motions" (vol. iii. p. 10).

Here at the fountain-head is found the idea of the nervous nature of the clinical phenomena of angina pectoris, a conception of the condition shared by many subsequent writers.

The next step worthy of note was made by Dr. Caleb Hillier Parry, a physician in Bath, who published in 1799 a monograph entitled *An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris.* This work is characterised by remarkable clinical acumen, and Parry's views, it seems to me, have not always been accurately presented by subsequent writers. Some of the clinical points dealt with by him are to be specially noted. He points out, for instance, that the first symptom is "an uneasy sensation (described variously as a stricture, an anxiety, or a pain) about the middle of the sternum, across the left breast, and in certain stages of the disorder usually stretching into the left arm" (p. 42), also that the pain occurs in paroxysms, and that early seizures seldom occurred without apparent cause (p. 43). He also insists, "we shall always find it (the pulse) become more or less feeble according to the violence of the paroxysm" (p. 45). Here we have an early recognition of great differences in the

degree of severity in the leading symptom. The pathology of the condition was held by him, and by his friend Jenner, to be disease of the coronary arteries interfering with the blood supply to the heart. His induction was that a heart with such coronary arteries could not bear any extra strain put upon it; that if subjected to extra strain it became surcharged with blood; that its action became enfeebled, and might cease. The extra strain could be induced by mental emotion, by physical effort, and by gastro-intestinal conditions. As the heart condition advanced, even slighter causes than these might produce paroxysms. Thus heart failure and its organic cause formed the central, and to him the essential, factors, in determining the gravity of the condition. According to Cullen's nosology, this was assuredly a *syncope*. "The motion of the heart diminished, or even for some time ceasing. *Motus cordis imminutus, vel aliquamdiu quiescens.*" It differed from an ordinary syncope "only in being preceded by an unusual degree of anxiety or pain in the region of the heart." He therefore termed the disorder *syncope anginosa*. To make the position of this old Bath physician still more clear, let me give his own words regarding a patient who had been long subject to attacks of what he considered a "very pure angina pectoris." He accompanied his patient on a walk uphill in order to witness what occurred during the fit. He says, "When the paroxysm was thus excited, I could perceive no symptom of disorder in addition to the uneasiness in the breast, except a gradual and most evident diminution in the strength of the pulse" (p. 59).

Allan Burns, Professor of Anatomy and Surgery in Glasgow, and practising in that city (1809), accepts Parry's view that syncope anginosa orginates "from some organic lesion of the nutrient vessels of the heart" (p. 137). He compares a heart with "cartilaginous or ossified" coronary vessels to a limb with a ligature round it called into active exercise. The limb so bound can only work for a very short time; "it soon fails and sinks into a state of quiescence" (p. 138). He holds that "the essence of this complaint consists in a reduction of the action of the heart" (p. 146).

In 1815, Jurine, in an essay to which the Academy of

Medicine awarded a medal, combated Jenner's and Parry's view that angina pectoris was due to ossification of the coronary arteries. To him "sans douleur sternale, il n'y a pas d'angine de poitrine" (p. 72), and he argued that the angina was essentially due to an affection of the pulmonary nerves (p. 123).

Laënnec, (1819), had found that in several subjects who had laboured under angina pectoris, and in whom there co-existed either hypertrophy or dilatation of the heart, there was no ossification of the coronary arteries (p. 757). According to him, "angina pectoris in a slight or middling degree is extremely common, and exists very frequently in persons who have no organic affection of the heart or large vessels" (p. 756). Desportes (1811) is quoted by Laënnec as holding similar views. Angina pectoris was to Laënnec a neuralgia.

Forbes (1832), the translator into English of Laënnec's great work, divided cases of angina pectoris into organic and functional. The pain to him was the result of "some unknown temporary condition of the nerves of the part," and he believed that "such a morbid condition of the nerves may be produced in a heart in all other respects sound."

Bertin (1833) also repudiated the view that angina pectoris was due to ossification of the coronary arteries. He had seen the symptoms "accompanied with a multitude of organic affections, either of the heart or of the large vessels, or even of the lungs" (p. 410).

Hope (1839) held that "the nature and variability of the symptoms of angina pectoris confirm the opinion of Laënnec that it is a neuralgic affection" (p. 499).

Latham (1846), along with a vivid portrayal of angina pectoris, considered the disease to be "a certain assemblage of symptoms, and not any constant pathological condition belonging to the structure of the organ" (p. 399). The paroxysm was to him "plainly a compound of pain and of something else" (p. 385). It was a "spasm of the heart," as it had been to Heberden. But then note his conception of spasm. He says: "Spasm is always accompanied with pain. And pain and spasm, wherever they are, disable the part which they befall" (p. 385). "In spasms of smaller

degree the heart fails to close freely upon the blood and to impel it freely into the arteries. In its spasm of greater degree it fails to project it altogether" (p. 386).

Walshe (1851) looked upon angina pectoris as a "paroxysmal neurosis in which the heart is essentially concerned" (p. 190). He thought genuine angina pectoris to be a very rare disease, while on the other hand he "very frequently met with a form of complaint combining in a minor degree many of the characters of angina." To this "imitation of the true disease" he gave the name of "pseudo-angina." He thought that this was the explanation of Laënnec's notion that the condition was of very frequent occurrence (p. 203–4).

Stokes (1854) agreed with Parry, and says "that the symptoms of angina arise from a temporary increase of weakness in an organ already weakened" (p. 486). He disputes Latham's idea of spasm, and thinks the idea of syncope more in accordance with observed fact.

Trousseau refers to the variety of pathological change which had been described as having been present in the hearts of persons who had suffered from angina pectoris. On the other hand, he has of necessity to admit that angina pectoris, even when most intense, need not be a symptom of an organic lesion. He therefore regarded the organic changes, when they were present, as non-essential, as coincidences, and held that the real affection was a neurosis—in fact, a neuralgia. That there might be no ambiguity in the minds of his hearers, he again repeated his opinion that it might be only "an idiopathic neuralgia."

Three Views.—At this point we may draw a line and say that up to it there were, if we exclude Jurine's view —which we may safely do—three views regarding angina pectoris: the first, that it was a convulsion of the heart, or a spasm of it; the second, that it was due to interference with its blood supply; and the third, that it was a neurosis—the word having no subtle or obscure significance, but meaning simply and plainly, a neuralgia—an idiopathic pain in nerves. Up to this time, it is also important to note that the new term was not limited to a relatively rare malady, characterised by a few dramatic incidents and a suddenly tragic conclusion. Its use had become so extended that

Walshe deemed it desirable to introduce the term pseudo-angina, under which to range the cases that, in spite of the correspondence in symptoms, had not the grave significance that the fatal angina of Heberden presented.

Angina Pectoris Vasomotoria.—With the year 1867 a new factor was introduced into the discussion on the nature of angina pectoris. Nothnagel in that year wrote a paper which he entitled *Angina Pectoris Vasomotoria*. In this condition the symptoms closely resembled those of angina pectoris, and would certainly, I think, have been placed under that designation by some of the distinguished clinicians whose works have been referred to. The essential point in this paper was the recognition of the important clinical fact that in this group of cases narrowing of the arteries preceded and caused the cardiac embarrassment and other symptoms; that, in fact, the anginous symptoms were secondary to the vasomotor ones; and that by relieving the cramp in the vessels the anginous symptoms were prevented coming on.

Earlier Observations on Blood Supply and Muscle Pain, **Intermittent Claudication.** — Before Nothnagel, Latham, when dealing with the treatment of persons the victims of angina pectoris, had not failed to note the guidance to be obtained from a true estimate of the condition of the vascular system. He says:

"The paroxysm is often put off and its severity mitigated and life prolonged by no means more surely than by keeping the vascular system in a just balance between fulness and emptiness, between rich blood and poor blood" (p. 405).

Three years before the date of the publication of Latham's lectures, Sir Benjamin C. Brodie's *Lectures on Pathology and Surgery* were published (1846). In them there is a lecture on senile gangrene which I doubt if any living surgeon could add to. He notes that the condition might be due to ossification of arteries or to obliteration without ossification. He enlarges upon the prodromal symptoms of gangrene—he had noted the numbness, pain, and loss of muscular power—which may precede its visible manifestation. With the clinical instinct of the great men of his time, he applied these observations to interpret the much-discussed question as to the nature and causes of the pain in angina. The

paroxysm of pain in angina varied in intensity, so it did in gangrene, greatly. The pain in gangrene might become excruciating—the pain of angina might fail to find words adequate to convey a true sense of its anguish. "The disease," he says, "is called neuralgia, which," he adds, "means nothing" (p. 356). A clear thinker, as well as a masterly observer, this surgeon of sixty years ago!

A still earlier recorded observation bearing upon the relation between lessened blood supply and pain must not be omitted were it only for the sake of historical accuracy. At the Académie Royale de Médecine, the *Séance* of 4th October 1831, Boullay submitted an observation he had made on a mare of a condition to which he gave the name of "intermittent claudication." The symptoms came on abruptly after some minutes' exercise, and they were found to be due to obliteration of the femoral arteries. When the animal was in repose the blood was able to reach the limbs by the collaterals, but when it trotted the compressed collaterals did not furnish enough blood, and the limbs were seized by *engourdissement* and by severe pain, which made the animal fall.

Charcot, twenty-six years later, revived this subject, and drew attention to a corresponding condition as it occurred in the limbs in man as causing severe pain, and as a premonitory sign indicative of a tendency to senile gangrene. So far as I have found, he did not apply his observations to the elucidation of the symptoms of angina pectoris as the English surgeon Brodie had done ten years earlier.

After this, however, intermittent claudication as a cause of painful muscle spasm took its place in medical literature, and has been much used by later writers when discussing the cause of the pain in anginous paroxysms.

Arterial Spasm.—Standing alone, and forming an epoch by itself, is Lauder Brunton's discovery (1867) that dilatation of the peripheral vessels by the inhalation of nitrite of amyl removed or relieved anginous symptoms. From this date vasomotor spasm became a still more definite factor in the consideration and the interpretation of the "syndrome of symptoms" known as angina pectoris.

What is included under "Angina Pectoris"?—What,

then, are we to regard as angina pectoris? Are we to confine the use of the term to that type of case described with such dramatic force by some of the earlier writers that there still lingers round its earlier records a glamour of picturesqueness? Are its symptoms to be bounded by the *dolor pectoris* and the *angor animi* of its early recognition, and is its close to be characterised by the same tragic suddenness? I think not. From the time of Parry the term "angina pectoris" has been applied to symptoms of less gravity, and to conditions where the outlook was not necessarily grave. Let me clinch this statement by quoting the words of Professor W. T. Gairdner, who, writing in 1877, says: "We now know that this typical angina is only the culminating form of a group of symptoms, which in their less pronounced, less definitely painful, and more complicated forms, are found to permeate the whole field of cardiac pathology and diagnosis" (p. 570).

From his own personal experience he says: "There is often an element of subjective abnormal sensation present in cardiac diseases which, when it is not localised through the coincidence of pain, is a specially indefinable and undescribable sensation" (p. 565). "A sensation which can only be called anxiety, or cardiac oppression" (p. 566).

To this group of symptoms he gave the special title *angina sine dolore*, recognising thereby what he believed to be "its true diagnostic and pathological significance and its alliance with the painful angina of Heberden." Even the dolor pectoris is, then, no longer an essential symptom; and the angor animi is whittled down to a sensation of anxiety. This shrewd clinical pathologist saw, however, that there were phenomena which, while different to, were of the same kind as those of the classical angina. This is what Walshe recognised when he used the much-abused term "pseudo-angina." While Gairdner places angina amongst the neuroses, he hardly, I think, attaches the same meaning to the word as the earlier writers did; and I am not sure that he does not somewhat grudgingly stick to the term. He was clearly impressed by the possible importance of vasomotor spasm.

Dr. Sansom, on the other hand (1892), thinks it probable

that a paroxysm of angina pectoris is an intense nerve storm, provoked by impulses conveyed to certain cerebro-spinal centres (p. 13). He refuses to adopt the theory of cardio-arterial spasm as the indispensable cause of the pain (p. 438).

Professor Osler, in his erudite lectures on the subject (1897), after defining angina pectoris, proceeds to say that the term is employed generically to define paroxysmal attacks of pain in the chest, qualifying the varieties by such names as true, false, hysterical, and vasomotor (p. 8).

Sir Douglas Powell, in a very admirable article, classifies angina pectoris under (1) a vasomotor group, and (2) angina pectoris gravior, which includes secondary and primary cardiac angina. Forbes, Bramwell, Gibson, Oliver, and others recognise two classes, organic and functional. Huchard insists that there is only one angina pectoris, and that it is always due to ischæmia of the heart muscle.

From all this it is quite clear that the term is no longer confined to the severe and fatal malady described by Heberden. It has been extended to a great variety of conditions, characterised by some sense of precordial discomfort, or of discomfort referred directly to the heart, which may or may not be accompanied by pain, and which may or may not be associated with coarse anatomical lesion. It was to cover these lesser manifestations that such terms as pseudo-angina, angina notha, false angina, and functional, as distinct from organic, have been used and applied.

Angina is indeed, to use Osler's words, "a syndrome or symptom group," the clinical pathology of which has to be determined in each separate instance. The term angina pectoris has in fact the same nosological significance as the term dropsy formerly had. It is not a disease; it is a symptom or a group of symptoms, and it is no longer the symptom but the cause behind and determining it which is the problem presented to the clinical pathologist of to-day.

Constructive and Critical.—In turning to the constructive side we have to look at certain vasomotor phenomena which are to be observed in the body; and I hope to make it clear what I regard to be the relations of those phenomena to the phenomena of angina pectoris.

The presence of arterial spasm has been recognised by

many who have written on angina pectoris since the date of Lauder Brunton's epoch-making discovery. Views as to the causation of the spasm have been various, and the relation of the spasm to the symptoms has also been variously interpreted.

An Arterial Abdominal Reflex.—We have to consider certain observations regarding an arterial reflex having its origin in the abdomen, the importance of which I think has not hitherto been fully appreciated. After taking food, and for at least part of the time during which the active processes of digestion are under way, there is an influx of blood to the whole splanchnic area, leading to hyperæmia, such a hyperæmia as considerably reduces the volume of blood in parts outside this area. This drainage into the abdominal vessels is balanced in the general circulation by a systemic arterial contraction. This is evidently a reflex phenomenon originating in the splanchnic system, passing to the vasomotor centre in the medulla, and thence transmitted to the systemic arteries. The existence of this set of phenomena has been carefully worked out by Dr. George Oliver in the average healthy person. The arterial contraction is shown by a definite diminution in the diameter of the vessels. It is to be regarded as a physiological hypertonic contraction. The fact, then, with which I begin is the change in the systemic arteries during active digestion—whether it be called narrowing, contraction, or hypertonus, signifies not, so long as the word we use expresses our thought. The change will be found to be a reduction in size and an apparent thickening of the arterial wall. The degree of these arterial changes depends upon the kind of meal which has been taken. In the big flesh-eater and the wine-drinker the arterial contraction is associated with a rise of blood-pressure and a true increase of arterial tension.

Putting extreme cases aside, the general statement will be found to be correct that narrowing of the arteries is present, but in many people, both middle-aged and old, I do not believe that there is any constant rise of blood-pressure along with it. The point is not a purely academic one, for the small and constricted vessel, with its slight

systolic expansion, is very commonly regarded as an indication of heart debility, and treated as such. In other cases the mistaking of hypertonus for a rise of blood-pressure may be of no practical consequence, for the treatment applicable to the one is suitable for the other.

The point here insisted on is the existence of this arrangement for adapting the circulation to the normal processes of ingestion and digestion of food. It is a normal reflex process. It is a systemic vessel constriction originating in a normal stimulus in the abdomen, and brought about through the anatomical connections in the nervous system. It is not, therefore, a "neurosis"; nor is the exaggeration of this reflex, prompted by a large meal of stimulating foods, a neurosis. It is necessary to lay emphasis on this distinction. This reflex, like all normal reflexes, is a beneficent arrangement, and could be easily shown to be one of the means of protecting the body from injury. It varies in delicacy in different persons; it exists in all.

Exaggeration of Normal Abdominal Reflex.—Granting the existence of this normal reflex, it will not be questioned that high feeding and alcoholic liquors accentuate it; and that under such circumstances there is marked raising of arterial tension in addition to arterial contraction.

It must, however, be here recognised that in the latter conditions there are two elements present—one, the vascular reflex; and second, the passage into the circulation of substances produced in the processes of digestion, and of substances derived from the alcoholic liquors drunk. It is not possible at present to give these two factors their precise place in bringing about the accentuation of vessel constriction and of stimulating the increased tension. The existence of the reflex is granted, and there is really little room for doubt that certain substances present in the blood directly stimulate vessel contraction. Contraction so induced is not a neurosis; it may, indeed, be induced when the connection with nerve centres is destroyed.

Action of Reflex on Sclerosed Arteries.—This recurring arterial contraction, especially if associated with increased arterial tension, is, as I pointed out some time ago, the efficient cause, and the usual cause, of arterio-sclerosis in the

sense in which this term ought to be used. At the same time I dwelt upon the importance of recognising that sclerosed vessels are not, because of the change in their walls, incapable of contraction, but that, on the contrary, they are prone to become hypertonic from causes which are commonly regarded as trifling. Professor Pal, as seen in his recently published work, *Gefässkrisen*, has also observed that sclerosed vessels are particularly sensitive, and specially liable to hypertonic contraction.

Let us bear in mind then—*First*, the existence of this systemic arterial reflex having its origin in the digestive organs or processes. *Secondly*, the influence of the character of the material introduced into the digestive system in accentuating the reflex. *Thirdly*, the effect of the reflex in producing arterio-sclerosis. *Fourthly*, that sclerosed vessels are particularly liable to hypertonic contraction.

Relation of Reflex to Angina Pectoris.—The relation between the phenomena just dealt with and angina pectoris is not at first sight apparent; and, in order to demonstrate the close connection between the two, I must now as briefly as possible refer to several illustrative cases which I have recently observed.

CASE 56.—*Angina pectoris gravior, showing extreme sensitiveness of arterio-cardiac reflex and its production by taking nourishment.*—The patient was a professional man, aged 67, whom I saw for the first time on 6th September 1904. He had suffered for years from definite and characteristic attacks of angina pectoris. Lately he had been much worse, and had been confined to bed for weeks. When I first saw him he was having many attacks daily, and circumstances of the most ordinary kind evidently brought them on—such circumstances as the taking of nourishment, the presence of flatulence, slight excitement, and even the changing of his position in bed. The severity of the attacks varied within considerable limits; the more severe, and therefore the more typical, attacks began with pain in the left elbow, which soon extended upwards to the shoulder and then downwards over the precordia. During an attack the hands became cold and painful. The medical attendant, who was

watching the patient with the greatest assiduity, informed me that the pulse always indicated the advent of an attack; the radial artery became more definite, firmer, and smaller; the pulse rate increased; while the wave became small, and might almost disappear. In some of the worst attacks the patient had become unconscious and was thought to be dying. The treatment followed was the administration of nitrite of amyl and nitroglycerine for the more pronounced of the attacks, and the relief afforded by them had always been prompt. Under the influence of one or other the pulse wave became larger, the vessel wall softer, the hands became warm, and the pain passed off. The patient took a remarkably calm and intelligent interest in his attacks, and the foregoing is gleaned from his own and the medical attendant's description. The diet for some time had consisted almost exclusively of raw eggs and weak broth, while sedatives of various kinds had been employed medicinally. The patient had always been a large flesh-eater. There was no albumen or sugar in the urine. The medical attendant had been impressed by the fact that the patient more recently almost always had a seizure after being given nourishment, even when that consisted of some raw egg. I had the opportunity of confirming the observations on the change in the pulse during a paroxysm soon after my arrival at the patient's bedside. During the time I spent talking quietly to him the pulse was small, soft, and feeble. I examined the heart without causing any discomfort. The sounds were so faint that they were occasionally inaudible, although the chest wall was thin. I next proceeded to examine the abdomen, and had my hand placed quite lightly on the epigastrium when the patient told us that an attack was coming on. I at once removed my hand. He took his attack with remarkable composure, and it was evidently not accompanied by the distressing sensations which so frequently accompany anginal seizures. Pain began in the left elbow, which, as has already been noted, was always the premonitory symptom of the more severe seizures, and extended to the shoulder and then to the precordia. The face became pale. The radial artery became quite abruptly hypertonic—that is, it became firmer and smaller, the rate rose to 100 and 110, and the

tension was sensibly raised. He was at once given a tabloid of nitroglycerine, and in a very short time the artery relaxed and the pulse became large, strong, and rather thumping, the pallor of the face was exchanged for a ruddy tint, and the pain ceased. The thumping soon passed off, and the pulse resumed the small, feeble character it had presented at the outset.

Comments.—There could, of course, be no doubt that the patient was the victim of a severe form of angina pectoris, and that the attacks were accompanied by marked arterial constriction. It was fortunately easy to arrest the seizures. They had, however, persisted for weeks without any lessening in their frequency or in their severity. The question which awaited solution was, Could anything be done to lessen the tendency to the attacks? Palliation was easily achieved; but could any measures be adopted which might be more curative? The point in the history which was most uncommon was the fact that the taking of nourishment was almost always followed by an attack, although the amount taken was small and could hardly be further reduced. Acting, however, on this indication, it was decided to materially alter the diet, the nature of which has been already stated. The dietary now advised was to consist of peptonised milk, peptonised oatmeal gruel, etc., and one raw egg well beaten up, daily. The patient was moreover given small doses of arsenic, tincture of strophanthus, and spirits of nitrous ether. This line of treatment was persisted in, and when I saw the patient a fortnight later the report of his condition showed considerable and satisfactory improvement, the attacks had greatly diminished in number and severity, and the taking of nourishment was only occasionally followed by an attack; the pulse was stronger. The tongue, however, had become coated, and small doses of rhubarb and soda were advised.

A week later the report was that the attacks had still further lessened in severity and frequency, and that when an attack was threatened it could be averted by a dose of aromatic spirit of ammonia or even by a dose of rhubarb and soda mixture. When pain in the elbow came on, it was accepted as an indication that the threatened attack would be somewhat severe, and the patient was at once

given a small dose of nitrite of amyl. The sensations which now more ordinarily preceded an attack were a sense of discomfort and distention in the stomach, and attacks were often associated with definite flatulent distension, the escape of gas being followed by relief. The medical attendant noted that when the abdominal discomfort supervened the pulse rate increased, and there was distinct hypertonus; as the attack passed the vessel wall relaxed and the rate returned to normal. Within the next two months the patient's condition varied a good deal, but it was noted that any gastro-intestinal or digestive upset at once threatened to induce an increased number of attacks. The management of the diet and of the digestive system required much tact and judgment, and was admirably conducted by the doctor in charge. In some weeks the patient had improved so much that he was able to be out of bed and to move about the house for the greater part of each day, and to interest himself in outside affairs. His clinical history for the succeeding months was chequered, for as his condition improved, it became impossible to continuously diet him as was desirable; still, at the end of June 1905 he had not had a seizure for six weeks, and was in fact freer of attacks than he had been for years.

Soon after this, however, albuminuria made its appearance with anasarca. The anasarca was got rid of, but the albuminuria persisted; Cheyne-Stokes breathing developed and became extreme. The patient finally developed gangrene of his entire right lower limb, of which he died in the end of October 1905, a proof that his arterial system was more degenerate than the radials indicated. I submit the case as illustrating an extreme exaggeration of the normal reflex which has been described. The angina in this patient was associated with a weak and somewhat enlarged heart. The hypertonic spasm of systemic arterioles, resulting from gastro-intestinal stimulus, proved so severe a strain on the weakened heart as frequently to lead to a syncope, during the anginal paroxysm, which was only short of being fatal. If we had had the opportunity of seeing the heart, it is probable that atheromatous or ossified coronary arteries would have explained its debility. The instructive feature

was the extraordinary sensitiveness of the peripheral organs from which the reflex started, and the success in allaying that sensitiveness by changing the kind of nourishment given, and its partial predigestion. The remedial effect was so pronounced that it gave me the impression that in this patient the normal reflex had been irritated beyond control by the free use of the more pure proteid foods, until it responded to the most trifling local irritation. That the anginous symptoms were always determined by the vascular hypertonic contraction was undoubted, and, so long as the latter could be restrained, there was no angina, and, as has been indicated, the key of the position was in the alimentary system.

CASE 57.—*Condition allied to angina, showing the stimulation of the arterio-cardiac reflex by digestive disturbance.*—This case might be classed as angina sine dolore, but I use it as an example of conditions allied to angina pectoris, in which hypersensitiveness of the abdominal reflex gave rise to phenomena of considerable interest and importance.

The patient, aged 68, was a member of one of the learned professions, and had spent many years in the tropics. I saw him in November 1904, during convalescence from an influenzal broncho-pneumonia, and was informed that on the afternoon of the previous day, between four and five o'clock, he had had a "fainting attack," in which his face became pale, he broke out into a cold perspiration, and the pulse was imperceptible. The nurse had been greatly alarmed, as she thought the patient was dying. He soon, however, rallied from this, and I saw him in the afternoon of the following day. It is unnecessary to enter into the details of this case at this stage, beyond saying that when the medical attendant and I inquired fully into his dietary, it was found to be very faulty, and he was forthwith put upon a comparatively austere regimen.

He had no similar attacks during the following days, but he often spoke of an attack as impending, and he apparently expected an attack about four o'clock in the afternoon. It was thought by his family and the nurse that this was pure nervousness, and an expression of the

pessimistic view he tended to take of his condition. Five days later I again saw the patient, and obtained from the nurse the statements on which the preceding account is based. I was waiting by the patient's bedside for his medical attendant to arrive, and thought the patient, who was sitting propped up in bed, looking well, for his colour was good. His pulse was 80, soft, small, and feeble. The respiration, however, showed a long deep breath often with a little sigh, followed by several quiet inspirations; it was distinctly "cerebral" in type, and it made me somewhat anxious. I asked the nurse to give him half an ounce of whisky in water, although he had had a couple of drachms shortly before, as I wanted to see the effect it would have upon the pulse and respiration. The effect was unexpectedly striking, and even alarming. In a few seconds the pulse altered greatly in character: the artery very plainly tightened up, becoming smaller and harder, and the rate varied within very wide limits. It was frequently 88 per minute, then it would run up to over 100, and again sometimes fall even to 40. Along with this sudden irregularity in the pulse rate there were equally pronounced respiratory and cerebral phenomena. Lying with his eyes shut, the patient seemed to fall asleep, and the breathing was so quiet and shallow that only on close attention could it be seen that he was breathing at all. Then he wakened up with a start and a groan, followed by some deep breaths. This series of phenomena went on with perfect regularity as long as he was not spoken to, each part of the cycle only lasting some seconds. When asked on awakening what was the matter, he explained that he had become unconscious, and had been dreaming that he was falling down a precipice, or passing through some equally unpleasant experience, often in company with some old friend long dead. He spoke of these attacks as "delirium," and insisted that the present was only an exaggeration of what he had experienced every afternoon, and to which reference has already been made. He could be kept awake by speaking to him, and he preferred being spoken to, because, as he said, it helped him to fight against the attacks. When he woke up from the nightmare his words were sometimes incoherent and unin-

telligible for a few seconds, but he rapidly regained complete self-possession.

This alarming and curious condition lasted about an hour. It had come on about three hours after lunch, which had consisted of a chop, spinach, and one potato. Some time after that he had had milk and hot water and two drachms of whisky in lieu of afternoon tea. When the patient's condition was as has been described, I asked the nurse to make some tea, and he was given two-thirds of a moderate-sized cupful of this. The attack had completely passed off by the time the doctor arrived; the pulse had returned to the condition it was in before the attack, the breathing was regular, and he was quite calm and self-possessed. When the next meal came he was quite able to feed himself. I had no doubt the patient's view was correct that the symptoms shown this particular afternoon were only an exaggeration of the afternoon symptoms which he had spoken of, but which no one with more understanding than a nurse had witnessed. The doctor had called at various hours for the purpose of observing the attacks, but the stimulus of his visit appeared to be sufficient to postpone them.

Comments.—The phenomena in this case were very striking, and the symptoms could only be regarded as indicating an anxious condition of matters. The fact that they threatened chiefly in the afternoon, and had so far lasted only for a comparatively short time each day, was reassuring; while the time of their occurrence suggested a connection between the symptoms and the stage of the digestion of the midday meal, which was the principal meal of the day. Suspicion was all the more strongly directed to this view by the fact, of which I had no doubt, that the attack I witnessed was precipitated or intensified by the half-ounce of whisky given at my suggestion. The immediateness of the effect was startling: it gave the impression of being produced the moment the draught reached the stomach. It was indeed impossible to escape from the conviction that the phenomena pointed to the production by the alcohol of a peripheral gastric irritation or stimulation, which precipitated or intensified a condition which was

already threatening. On this assumption the local irritation must have been followed by a very prompt reflex effect. The impulse communicated by the sensory nerves of the stomach to the vasomotor centre must have stimulated that centre so as to increase its vaso-constrictor action, the result being the hypertonic contraction of the radial artery which was so markedly present. This stimulation of the vaso-motor centre must have also told in some way upon the cerebral blood supply, judging from the cerebral manifestations which were present. That even the motor area of the brain was involved was shown by the occurrence of muscular twitching and starting of the limbs, especially on the right side, when the other cerebral manifestations were at their maximum. The respiratory centre, as shown by the great respiratory irregularity, shared in the perturbation. This association, however, is of common occurrence. That the attack should have passed off as it did, without any therapeutic measures being taken beyond the administration of a few ounces of tea infusion, only strengthened the view that it had been determined by peripheral conditions of a temporary kind; that, indeed, the true cause of the trouble lay in an error in the digestive process at this particular period after the midday meal.

The diet was altered in some of its details, and for an afternoon or two the patient was given a dose of one of the commonly used vaso-dilators as soon as the symptoms threatened to come on. He had no more attacks; his recovery was complete, and he returned to his duties in the East. Nearly three years later there had been no recurrence of the symptoms.

CASE 58.—*Anginous paroxysms first appearing on physical effort: influence of emotion and of diet.*—This patient was a public servant, whom I saw in July 1905. He had suffered for some time from attacks of typical angina pectoris, commencing with pain referred to the heart. This symptom first appeared when he was hunting, and so constantly reappeared when he attempted to follow the hounds that he had to stop doing so. He had had one or two attacks of great severity during the night, in at least

one of which it was thought he was to die. He had been treated with considerable success by an eminent Irish physician. He carried about tabloids of trinitrin in his waistcoat pocket, and had been taking potassium iodide for some time. His condition had become practically stationary: hunting was out of the question; he could only walk on level ground; the slightest hill, or the slightest hurry, produced a breast pang, which arrested further activity. When I first saw him he was having a slight attack almost nightly, soon after midnight, and at that time he usually took a tabloid of trinitrin. He claimed to be a small eater, and his daily wine consumption did not exceed two or three glasses. His dietetic habits had not been seriously inquired into, and had not been altered. He had been told that the artery of his heart was rigid, and he and his family were dwelling under the shadow of the fear of sudden death at any moment. On examining him I found that the heart was slightly enlarged, the dulness reaching to the nipple line, but the sounds were clear and pure at apex and base. The pulse was regular, of good strength, and gave no indication of an enfeebled myocardium. The vessel wall was much thickened for a man of his age, and in view of the arduous life he had led at home and abroad; there was no albumen or sugar in the urine, but it tended to be scanty and high-coloured. The bowels were acting, but not freely, and his conjunctivæ indicated "sluggish liver." The question here, as in Case I., was whether anything could be done to carry improvement beyond the point it had reached. I was again fortunate in witnessing a slight attack in this patient. When I was examining into the state of his heart and vessels, which under the circumstances I was doing with great care, and not hurriedly, he became a little agitated, and informed me that he was having a slight attack, with the usual feeling in his heart. On putting my finger on the radial pulse, it had become markedly hypertonic. In a very short time the hypertonic spasm relaxed, and the heart sensation vanished.

Here was another instance of the pain and anxiety of angina, assuredly due to arterial spasm. There seemed to me, further, no doubt that the measure of emotional dis-

turbance aroused by the medical examination had induced the attack. There was, therefore, no doubt as to the existence of marked vasomotor sensitiveness. As the patient was already taking potassium iodide more or less regularly, and trinitrin when threatened with a paroxysm, I felt that not much benefit could be expected from further medicinal measures. Guided again, therefore, by the theory that this vasomotor sensitiveness owed its origin to the circumstances and conditions of the alimentary system, I strongly advised that all wine should be stopped, and that other changes be made in the diet. The progress of the case need not be enlarged upon; it is sufficient to say that the patient made marked improvement in every respect. Two years later this patient was reported as in good health.

CASE 59.—*Angina with gastric disorder: attack caused by palpation of epigastrium.*—This was a female patient in the Royal Infirmary, who complained of attacks of pain in the chest, over the precordia, with shortness of breath and of indigestion and pain in the epigastrium. The heart was weak and somewhat dilated. I mention this case on account of an experience I had in connection with it somewhat resembling an incident noted in Case I. On examining the patient's abdomen I found that there was surface hyperæsthesia in the epigastrium, as is common in some forms of gastric disturbance; and as I was gently palpating the region, I noticed the patient's face become pale and expressive of anxiety and discomfort. I at once put my finger on the pulse, and asked her what was the matter; she replied that the pain in her chest had come on. The radial artery had become markedly contracted and hardened. As the hypertonic spasm of the artery relaxed the heart discomfort passed off.

Clinical Pathology. — By gathering up the separate points, we are able to explain the whole clinical pathology of angina pectoris. In the first place, there may or may not be a morbid anatomy. When present, it varies so widely in different instances that, from the earliest times to the present, many physicians have refused to accept any anatomical change as sufficient to explain the phenomena. Whenever this is

the state of knowledge, it will be found that men turn to that scapegoat of all that is unknown—the nervous system.

In attempting to interpret the phenomena of angina pectoris, I shall begin with an illustration of the simplest form of angina. Tobacco poisoning, from smoking, is commonly recognised as the cause of a form of angina pectoris—a toxic angina. It so happens that I can give myself tobacco angina by smoking strong tobacco. The symptom which arouses me to the fact that I must put my pipe aside is a sense of constriction at the heart, a feeling as if it were grasped; it is not a very unpleasant sensation, but it gives one the impression that it only required to be much intensified to make the dolor pectoris to be avoided, whatever self-denial it required. The heart discomfort is accompanied by a hypertonic contraction of the radial arteries. Discomfort and hypertonus disappear together. If I am told this is a neurosis, all I can reply is, if it is, opium poisoning, or tetanus, is also a neurosis; and that I do not look at morbid processes from that standpoint.

Whatever the degree of intensity the heart-pang may reach, it is, I believe, always the result of a *sudden* embarrassment of the myocardium, and usually and chiefly that of the left ventricle. This sudden heart embarrassment, varying widely in degree and intensity, is the one fact common to all the divisions or subdivisions under which all cases have been classed. The *angor animi*, which also varies in degree, is, I think, common to all sudden heart embarrassments, which fall short of being so immediately fatal that there is no time for the development of such sensations.

Professor Clifford Allbutt attributes the pain to an aortitis or a periaortitis. When either of these is present, the raising of aortic pressure following upon arterial contraction may well cause a pain comparable to pain caused as I have just indicated.

The heart embarrassment is, as Mackenzie has quite recently pointed out, an "impairment of the function of contractility." This is the modern expression of what Parry meant by "syncope." It is to this condition, associated with pain, that Rosenbach confines the term "stenocardia."

The impaired contractility is due either, in the first place, to the sudden strain put upon a feeble myocardium by general arterial contraction, or by a call for increased work; or, in the second place, to a myocardium feeble, not from anatomical changes in it, but temporarily so, from deficient blood supply, the result of the participation of the coronary arteries in a general arterial spasm.

The cause of the sudden heart embarrassment in the vast majority of cases is the arterial hypertonic contraction, or spasm contraction, we have been considering; the latter term indicating the more sudden and severe degrees of contraction. Even toxic conditions only give rise to angina when the factors indicated are present.

By means of the cases reported I have endeavoured to illustrate the remarkable relations which exist between the digestive system, or the materials introduced into it, and the general arterial system. I have also shown that conditions having their origin in the abdomen can induce a hypersensitiveness of a normal vasomotor reflex, which becomes apparent as arterial spasm, whenever the exciting conditions are sufficiently pronounced; and that when those conditions are altered the arterial spasm disappears.

As I have already indicated, the arterial spasm may be determined by direct irritation of the stomach. In two of the preceding cases the spasm and its accompanying paroxysm of angina were caused by palpation of the epigastrium. This illustrates the well-known nerve connection between surface and viscera—gastric disorders frequently causing surface hyperæsthesia, while in these two cases the surface stimulus produced the same effect as if a stimulus had been applied directly to that viscus.

The stimulus is also provided by the substances taken into or absorbed from the alimentary tract. These substances exercise a supreme influence in producing and in maintaining the hypersensitiveness of the vasomotor reflex, and therefore of the vasomotor centre.

This *hypersensitiveness of the vasomotor centre* explains what has long been recognised—that paroxysms of angina have as their main determining cause physical effort, mental emotion, or digestive disturbance.

The Unifying Principle.—By the application of the facts I have brought under review the group of symptoms known as angina pectoris can be satisfactorily explained, no matter how diverse the conditions may be in which it occurs. It explains the occurrence of the symptoms without anatomical change, while at the same time it shows the importance of the presence of anatomical changes in the coronary arteries, or in the myocardium. In this connection it must always be borne in mind that even calcareous coronary arteries are not calcareous to their finer subdivisions, so that in this condition, as well as in the varying degrees of arterio-sclerosis, the power of spasm contraction is not lost—may indeed be intensified. It gives to the nervous system its due place in the production of the phenomena.

To illustrate the production of a paroxysm by mental emotion or physical effort, let me refer to Case 58, in which slight emotional disturbance or slight exertion had the effect of inducing a paroxysm. The connection between the emotional centres and the vasomotor centre is a normal one, and constantly active. In this case a normal emotional impulse acted upon the hypersensitive vasomotor centre, resulting in the arterio-cardiac phenomena of angina. This was no abnormal excitation of any emotional centre. It was a normal excitation passing on to an abnormally sensitive vasomotor centre. The vasomotor centre in this same patient was equally influenced by slight physical effort; it was not the voluntary muscle centres, nor the nerve endings in the muscles, which were unduly sensitive, but the vasomotor centre to the normal influences resulting from voluntary muscular effort.

By the cases reported I have also shown that this hypersensitiveness of the vasomotor centre, even in grave angina pectoris, can be reduced, controlled, or even removed by dietetic measures, with the result that the anginous seizures are removed or greatly modified. In cases where the arterial spasm is associated with great anatomical change, either in the myocardium or in the coronary vessels, absolute cure can hardly be looked for; but in all cases the symptoms of angina pectoris may be much ameliorated by accepting, as a working hypothesis, the propositions I have ventured to

submit, and conducting the treatment in accordance with what they indicate.

There was need of a principle which, when applied, would explain the phenomena in each of the various groups into which the disorder has been divided. There was abundant clinical experience showing the remedial effect on the paroxysm of vaso-dilators; but the only explanation of the arterial spasm which up to the present time has been offered has been, to use Sir R. Douglas Powell's words, that of "a pure neurosis of the cardio-vascular system."

Owing to the varying degree of intensity of the symptoms, I would suggest that in classifying the cases the simplest distinction might be found in the terms "angina pectoris major" and "angina pectoris minor," the former being confined to those cases in which there was believed to be permanent anatomical change in the heart or its vessels.

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CHAPTER XVI

THE PHENOMENA CAUSED BY HYPERTONIC CONTRACTION OR SPASM CONSTRICTION OF CEREBRAL ARTERIES

ALTHOUGH physiologists have not located a vasomotor centre for the cerebral vessels, there are many nervous phenomena which can only be satisfactorily explained on the assumption that these vessels can and do contract. To the clinical pathologist the hypothesis that tubes possessed of a muscular coat do not respond to stimuli, is as improbable when it is applied to the cerebral vessels, as it would be if applied to any similarly constituted structure in any other part of the body. Clinical medicine requires the recognition of such contraction. In such circumstances it is usually found that laboratory investigation ultimately is able to demonstrate what has been inferred from clinical phenomena. It is, indeed, desirable to keep the clinical aspects of various problems clearly defined, so that our physiological coadjutors may thereby be encouraged to continue their very important investigations, and perhaps be prevented from regarding questions as settled when clinical requirements are not satisfied.

It is important and interesting, however, to remember that all the arteries in the body are not connected with the vasomotor centre in the medulla. The systemic vessels and the splanchnic system are linked to this centre, and through it have mutual relations with each other. The vessels in the brain, the coronary arteries, and the pulmonary vessels are apparently not linked to it. In this chapter attention is confined to the first of these, and various clinical phenomena, common in clinical experience, are dealt with. The more severe of these are due to softening from permanent cutting

off of the blood supply from portions of brain, while the less severe are unquestionably due to interference with brain function, caused, I believe, by local disturbances or alterations in blood supply dependent upon temporary and evanescent vessel changes, and in a considerable degree capable of being influenced by therapeutic measures. A gradual intensification in the phenomena referred to will be found, rising from transitory sensations of numbness or tingling to hemiplegia and aphasia. Many of these phenomena have been entirely misunderstood, because the explanation of them had not got beyond the point of referring them to the vague category of the "neuroses." Any suggestion that the phenomena might be vascular in origin was limited by the tendency or habit of thinking that all vessel phenomena in the brain were embraced under either embolism, thrombosis, or hæmorrhage. The object of this chapter is to show more fully than I have yet done, that contraction of cerebral vessels is the direct or indirect cause of the phenomena referred to. I have dealt with this subject in papers already published, and since then it is to be noted that a few other writers have adopted a like view. In previous contributions to the subject of hypertonic contraction of cerebral vessels, I have dwelt in the main on hypertonus as shown in sclerosed vessels; that is, in cases where cerebral manifestations were associated with decided changes in the systemic vessels. Here I do not confine myself to this phase, but cover a wider field, so that the links in the chain of evidence may be more clearly seen to be united.

CEREBRAL VESSELS, ALTHOUGH NOT CONTROLLED FROM THE VASOMOTOR CENTRE, POSSESS CONTRACTILITY, AND RESPOND TO NERVE STIMULI

Physiologists have shown that the cerebral vessels are not under the control of the vasomotor centre in the medulla, and, as has been stated above, they have not so far found a centre for these vessels in any other part of the encephalon. It would, however, be very unsound to assume from this that cerebral vessels have little if any contractility.

It is of great interest, however, to realise the significance

of the fact that, while the great systemic system of vessels is under the control of the vasomotor centre, some local systems, such as the brain, are not so controlled. It was presumably this fact, coupled at one time with the failure to demonstrate nerve filaments in connection with cerebral and other vessels, which led to the hasty conclusion that the muscular tunic of such vessels was unimportant, if not even non-essential or unnecessary. The acceptance by the physiologists of the existence of nerve fibres connected with cerebral vessels entirely changes the standpoint. Both Morrison and Gulland claim to have demonstrated, by the newer methods of histological investigation, the existence of such filaments; and this has been accepted by so eminent a physiologist as Dr. Leonard Hill, who, while stating at page 146, vol. ii., of Schäfer's *Physiology* (1900), that "no evidence has been found of the existence of vasomotor nerves," has added a *note* at page 168, presumably as the work was going through the press, granting the existence of such cerebral vasomotor nerves. This being so, it is not necessary to labour the point that cerebral vessels contract and dilate, for the possession of a muscular coat and nerves in their walls settles that. The relations of these nerves have not, however, been demonstrated; but, notwithstanding this hiatus, it may be confidently assumed that nerve filaments, here as elsewhere, have central connections. Although these filaments are not connected with the systemic vasomotor centre, they must have connections with other centres; and it is probable, indeed certain, that some of these are with emotional centres. It is accepted that emotion influences cerebral function in other than emotional centres, and it is assumed from analogy that it does so by influencing local blood supply. It is not necessary to argue that local function and local blood supply go hand in hand. It is equally unnecessary to argue the proposition that local variations in blood supply must occur in the brain; and may evidently be determined by causes not acting upon the ordinary vasomotor centre, and therefore showing no similar or corresponding changes in the systemic vascular area controlled from that centre. While this is true, the converse must be equally true, namely, that conditions which act *through* the ordinary vasomotor centre do *not* act

upon the cerebral circulation. When the point is thus stated, and we allow our minds to look at it, and to realise that the separation of the brain blood supply from the influence of the ordinary vasomotor centre must protect the brain from a multitude of influences which would keep it in a constant state of instability, the arrangement becomes a very striking and evidently a beneficent one. That the cerebral vessels must have a vasomotor centre of their own seems, however, to be a reasonable inference, and perhaps the physiologists may yet be able to locate it. Meanwhile, we may safely assume that the cerebral vessels contract and dilate under the influence of certain nervous impressions or stimuli.

THE VESSELS ACTED UPON BY SUBSTANCES IN THE BLOOD.

In a previous chapter it has been pointed out that a number of substances used therapeutically act directly upon the vessel wall,—that is, without the medium of the nervous mechanism,—leading to contraction or relaxation of its muscular coat. The *direct* action of the secretions of the adrenal and pituitary glands upon the vessel wall has also been referred to as adding weight to this important and far-reaching proposition. That the secretions of these glands must take an important part in determining vessel tone, which really means the degree of contraction of the muscular tunic, seems to be an unavoidable inference, and if this be correct it requires to be further realised that the substances in question are probably present in relatively small amount in the blood. It is unthinkable that organs secreting substances with such striking properties are not in definite, important, and constant relations with normal and ordinary physiological everyday processes. It must, it seems to me, be accepted as a law that vessels respond directly to the influence of substances present in the circulating blood even in small quantity.

THE APPLICATION OF THE FOREGOING TO THE CEREBRAL VESSELS.

From the foregoing it is evident that we are entitled to formulate and to hold the following four important pro-

positions, namely: *first*, that vessels possessing a muscular coat must be regarded as capable of contracting and dilating, although not connected with the systemic vasomotor centre; *second*, that it has been experimentally proved that the contraction of vessels may be effected by the direct action on their walls of substances circulating in them, acting without the intervention of the nervous mechanism, and present in small quantity; *third*, that the phenomenon of vessel constriction is not necessarily present in all parts of the body simultaneously, but can be manifested locally; and *fourth*, that these propositions must be applicable to the cerebral vessels.

The two influences which affect the degree of tone, contraction, or constriction of vessels are therefore nervous influences and blood composition; and this must be true of the cerebral as it is of the systemic vessels.

Nerve Influence.—The effect of nerve influences is seen in the result of emotion on cerebral activity—it may exalt or depress, it may open wide the gates of speech, or it may cause a temporary loss of utterance. All similar and allied phenomena must be held as associated with focal modifications of blood supply, and consequently with a degree of local vessel constriction. This is our conception of brain function and its attendant blood supply. I need only quote one readily available physiological support to this, which states that in the cerebral vessels "there is to be recognised a movement of vascular elevation and depression occurring from twice to six times in a minute, corresponding to the periodic-regulatory dilatation and contraction of the vessels." It is further stated that "this movement is influenced by emotional disturbances."

Influence of Blood Composition.—The direct influence on the vessel wall of substances circulating in the blood has been so fully established, that it is only necessary to lay emphasis on the statement that the facts must be applicable to the cerebral vessels. Whatever difficulty there may have been in the past regarding the cerebral vessels in relation to nerve impulses, it is impossible to exclude the cerebral, or any other group of vessels, from the influence of blood composition. And I venture to suggest that it has been the

non-recognition of this fact which has given rise to inaccurate and misleading teaching.

Effect of Strychnine.—There is, it seems to me, a large and important field in connection with the cerebral vessels ready for investigation. As warranting this statement, I may, using the authority above referred to, point out that strychnine irritates the systemic vasomotor centre leading to constriction of vessels, while there is at the same time an increase in the amount of blood in the arteries of the central nervous system. This shows that the latter do not participate in the contraction; that, in fact, this substance has a different action upon the cerebral vessels to what it has on the systemic vessels, presumably because it acts through the systemic vasomotor centre, and not directly on vessel walls.

The Relation of the Pituitary Body to the Cerebral circulation.—This field for investigation seems to me to be even more interesting than the foregoing suggests. It has been shown that the hypophysis of the pituitary body has much the same constrictor influence on the vessels as the adrenal. It seems to me unreasonable to regard the pituitary hypophysis as a mere supernumerary of the adrenals, and yet I have not seen it suggested that the former may have any special relationship to the cerebral circulation. And yet what would be more reasonable or more in conformity with much of our more recently acquired knowledge of the relation of structure to function? Seeing that the cerebral circulation has a considerable measure of local autonomy, it is but reasonable to expect that this special structure is present where it is to facilitate the working, or even as necessary to the realisation of this autonomy. Although both glands have a similar action, reference has already been made to Haynes' work, which shows that there is a difference between them which is both interesting and suggestive. In the series of experiments with ergot, dealt with in a previous chapter, he has shown that large doses of ergot (which have the reverse effect of small doses) abolish the action of adrenal extract, while they do not so act upon pituitary extract. Large doses of ergot also lead to loss of sympathetic nerve action, so that ergot has a corresponding effect upon sympathetic and adrenalin action. It has, however, no such

action upon the pituitary, the cerebral analogue of the adrenal; and the cerebral circulation is also outside the systemic vasomotor system. The conditions which regulate the activity of the pituitary are as little known as those which determine adrenal activity, but it will not be questioned that Haynes' findings are highly suggestive, and suggest subtleties the investigation of which by physiologists might place most valuable measures in the hands of the clinician. Garnier and Thaon have shown that when the vagus is cut the pituitary hypophysis has no effect on the blood-pressure.

Clinical Application.—It appears from the foregoing that we are abundantly justified in requisitioning cerebral vessel contraction for clinical purposes, and also in holding that the state of the systemic is in some cases a guide to the state of the cerebral vessels, while in other cases it is not a guide. This being the position, it must be further assumed that the vessels in localised and circumscribed areas of brain can be affected without a like and simultaneous involvement of the entire intracranial circulation. Such a proposition is, in view of the anatomical and physical conditions, incapable of ocular demonstration; but even here we are not without cognate facts which warrant us in accepting it. It is well known that local vessel constriction is an accompaniment of migraine, and that relief of suffering accompanies vessel relaxation. It has also been noted that spasm of retinal vessels may occur in Raynaud's disease. Dr. Lundie recorded a case recently in which he contended that temporary blindness was due to spasm of retinal vessels, the blindness passing off when the constricted vessels relaxed. In the argument in support of his contention regarding the cause of the temporary blindness in his own patient, he refers to other observations of the same kind in which both generalised and limited constriction of retinal vessels had been noted. When the constriction is limited, the area of blindness corresponds with the position of the constricted branch. Lundie's interesting paper brings together sufficient facts to prove that temporary constriction may occur in all or some of the branches of the retinal artery, and my contention has been that corresponding processes occur in the brain. That

they occur in the retinal vessels gives strong support to my contention, and I hold may be claimed to be as near absolute proof as is attainable.

I have arranged the cases showing symptoms which I hold to be the outcome of vascular disturbance in the brain, into groups illustrating the degrees of importance of the symptoms. The groups also show a gradation of links which strengthens the contention that the cases have the same underlying clinical pathology.

The patients, showing the symptoms referred to, can be divided into two main classes; namely, those whose vessels were sclerosed, and those whose vessels were not permanently thickened. In the different groups both classes may be included.

GROUP I.—TRANSITORY PHENOMENA: SLIGHT AND GRAVE.

I begin with the cases showing minor and transitory phenomena, minor when occurring in persons with soft vessels; of much more grave significance when occurring in persons with thickened vessels. In the second class the symptoms are commonly and justly regarded as *premonitory of apoplexy.*

Accompaniments of Migraine.—It is not necessary to dwell upon the association of migraine with arterial contraction, for it is widely known and recognised. Not only that, but Thoma states that he has seen a case of supra-orbital neuralgia followed by thickening of the temporal artery, on the side attacked by the pain, the result of the recurring spasm. It is also well known that various visual phenomena may be associated with an attack, the most striking being a homonymous hemianopsia on one or other side. When recently talking over this aspect of my subject with Mr. R. Marcus Gunn, F.R.C.S., Senior Ophthalmic Surgeon at Moorfields Hospital, London, he informed me that he knew of cases of migraine which were accompanied with various defects of speech, such as partial or complete motor aphasia, a measure of amnesia, or a degree of paraphasia. He also knew of sensory phenomena referred to one side of the body,

of hemiparesis, or of brachial monoparesis. These various accompaniments coincided with the attack of migraine, and disappeared with its subsidence. Assuming the view of the relation between migraine and local arterial spasm to be correct, and personally I do not doubt it, it is legitimate to infer that the additional phenomena are the result of a corresponding spasm in the areas of brain to which they are referable.

Cases illustrating Transitory Phenomena.

CASE 60 was a young medical graduate, who informed me that when he was working hard and much run down in condition he had an attack of complete loss of speech and of power in the right arm. The condition after lasting for an hour passed off. He was subject to attacks of migraine, and it was thought that this seizure was a "migrainous manifestation." He himself, in the light of my published views on hypertonic contraction of arteries, had come to regard the attack as illustrating and confirming my contentions. This case corresponds with the cases referred to in the preceding paragraph.

CASE 61 was a medical friend in middle life, who on getting out of bed one morning had a sensation of numbness on the left side of his body, including his face. He could move his limbs and stand, but his limbs felt as if they did not belong to him. The attack passed off in a few minutes. The arteries were soft.

CASE 62.—A gentleman, æt. 69, consulted me regarding a recurring feeling of numbness in the right arm, which came and went without any cause so far as he could see. He had a feeble circulation, and a tendency to slight hypertonic contraction of his vessels. I gave him a pill containing iron and digitalis, and a mixture containing 10 minims of sp. etheris nitrosi in each dose, with instructions to take a dose whenever he felt the sensation referred to. He was quite confident that the ether gave him speedy relief, and he soon ceased to experience the unpleasant sensation mentioned.

CASE 63.—A man, æt. 63, consulted me at the Royal Infirmary on Wednesday, 30th May 1906. He stated that on the previous Saturday he completely lost the power of speech for an hour or two, but regained it fully before night. When he awoke on Sunday morning his whole left side was powerless, and his face was twisted to the right. Power had returned to the arm and leg sufficiently to permit him to walk to the Infirmary. When he presented himself there, he still showed a little feebleness of the limbs on the left side, and the face was drawn to the right side. There was no loss of sensibility. The radial arteries were markedly thickened, but there was no albumin in the urine. There had been no loss of consciousness.

CASE 64.—A man, æt. 48, was seen by me on Monday, 31st December 1906. His statement was that on Saturday morning about 8.45, when lying in bed before having breakfast, but an hour after having had a cup of tea, a feeling as of pins and needles began in his left ankle, spread up his leg, the left half of his trunk to the shoulder, and down the left arm. It was not in the neck or face. His arm became "cold and useless": he could move the arm but it felt useless, and he could not grip the bedclothes with his hand. He did not try his leg. In about half an hour this passed off slowly, and he "felt the heat gradually coming back." On Sunday morning (the day before I saw him) about a quarter to nine he had begun breakfast in bed and wanted to speak to his wife, but could not speak, "could only make a noise," and "get no words out." He stopped taking breakfast and lay quiet. About 10.30 speech returned, but it was "kind of stumbling" all day—"like a man that stutters." On Monday morning he was all right. He had been confined to the house from the preceding Monday with "a cold" (influenza). There was no albumin in the urine. The radials were slightly hypertonic, the pulse was feeble, and the heart sounds faint. He had some other attacks and came into my ward in the Royal Infirmary on 7th January. He was kept in bed and given liquor strychninæ and tincture of squill. He had three attacks of a "prickly feeling" in his left side, and one which he described as a "dragging," "like leather which

had been wet and then was drying." The pulse ranged between 78 and 84 or so. The hæmomanometer on three different days gave readings of 105°–95° and 100°. The pulse was soft and feeble. He went home on the 15th, and reported himself at the ward on the 19th, and was advised to continue his medicine.

CASE 65.—Thomas N., æt. 68, army pensioner, stated that on Saturday, 15th December 1906, he had been out walking about as usual. On the following day when he got out of bed he had a feeling of numbness in the left side of his face and the left arm and leg. I saw him at Queensberry House on the following Wednesday, the 19th, when he complained of a feeling of numbness and "stiffness" in his whole left side, and thought it was "rheumatic" and due to a "heavy cold." The condition had become worse since Sunday, and he felt that he was dragging the left leg more. He could move the left arm and hand, but he could not grasp my hand; he could just close his fingers on my hand. He had been moving about since Sunday, but he dragged the left leg a little. The face was drawn slightly to the right, there being distinct paresis of the left. Speech was not affected, but he became emotional when I told him he must go to bed. I sent him to my ward in the Infirmary. When examined in bed, in addition to the foregoing facts, it was found that sensibility to pinching was diminished and delayed on the left side. The knee jerk was absent on both sides. The plantar reflexes were not elicited by tickling the soles, but on deep pressure the right was much more active than the left. There was no Babinsky. The pulse was 66, the artery somewhat thickened, Oliver's hæmomanometer recorded a pressure of 165. He was given half a grain of erythrol tetranitrate every four hours. On the 20th, he was given, in addition, 5 minims tinct. digitalis every four hours, which was reduced to 3 minims on the 21st, as it had tightened his vessels; and as this effect was still more marked on the 22nd, it was stopped, and the erythrol was given every three hours, and in addition 5 grains of potassium iodide were given three times a day. On the 21st, the patient could draw up and extend the leg against considerable obstruction; no response

to tickling the soles; deep pressure gave marked reaction on right side, but very faint on left. On the 22nd, the grasp of the hand was a little stronger. On the 26th, the grasp of the hand had further increased. On the 27th, erythrol was stopped, iodide of potassium was continued, and he was given in addition 5 minims of liq. strychninæ hydrochloridi three times a day. On the 30th, he moved his left hand and arm freely, and said that they felt "quite well": the grasp of the hand was strong. There was still paresis of the left side of the face. The reflex to tickling the soles was prompt and equal on the two sides. There was no dragging of the foot when walking. The radial artery on the 29th, as well as on the 30th, was soft, all trace of thickening having disappeared. There was more colour in the face: at first his face was pale and somewhat pinched, now it was slightly ruddy. The hæmomanometer gave a reading of 120. He was allowed to go out on the 31st. I saw him on the 18th of January, when he confidently asserted that his arm and leg were "all right," but the left side of his face had not made any further measure of recovery, and he complained of his food gathering between his cheek and gum on that side. This man had remained well when this went to press in the middle of September 1907.

Remarks.—When the subsequent groups of cases are followed, it will be seen that the cases in this group divide themselves into the two classes already indicated,—the less grave cases being represented by the victims of migraine, in whom the symptoms are usually well under medical control, and the prognosis correspondingly satisfactory. The grave cases are those in which the symptoms are associated with vessel thickening, or occur in old people with soft vessels and a feebly acting heart. The gravity of these latter cases lies in the risk of cerebral softening taking place, the steps which lead to such a disaster are discussed later.

These cases lead to a second group in which there was recurring mental or motor phenomena. In this group there is probably always arterial disease.

GROUP II.—RECURRING MENTAL OR MOTOR PHENOMENA.

In this group I include a number of cases, which illustrate the considerable variety of clinical phenomena which may be encountered, and the significance of which it is desirable not to misapprehend in practice. I begin the group with a retired military man.

CASE 66, æt. 70 years, had thickened arteries, and marked arcus senilis. There was no albumin in the urine. He complained of having become unusually somnolent after lunch and dinner; his speech had become a little thick and blurred; but what annoyed him most was that he had begun to spell words incorrectly in his letters, a thing he had never done before; his gait had also become slightly unsteady. In fact, his condition at the time to which I refer, which was some years before he died, was such as to suggest rapid cerebral degeneration. I was, however, satisfied that the symptoms were due to hypertonus of his sclerosed vessels curtailing the brain blood supply. I knew that in eating and drinking he was very moderate, as judged by ordinary standards, yet after careful consideration I strongly advised him to give up the glass of claret he took at lunch and dinner. He at once fell in with my advice; and was rewarded by the disappearance in a very short time of the symptoms which had given us so much concern. Along with the improvement there was a definite arterial relaxation. I mention this case because it is illustrative of a type. There was a very marked readiness on the part of the arteries to tighten up a little, and for long periods a small quantity of potassium iodide was sufficient to control it. After the continuance of such a period there would be some deviation from the ordinary austerity, some extra proteid added to the dietary, or some small measure of alcoholic fluid taken at lunch or dinner, and as sure as this occurred there was a threatening of the former symptoms, and an appreciable hypertonic tightening up of his radial arteries. The total quantity of proteid—and, indeed, of every kind of food taken—was relatively small; and the same was true of the amount of wine or of spirit. A more regular and temperate

man could hardly be found, and yet he was constantly threatened with dangers more associated with the opposite habit. How is a patient of this kind to be looked at? Are we to be content to regard him, as is not unusual, merely as an old man whose "strength" has to be kept up? This view, when acted upon, ends speedily in disaster. I submit that this man showed in later life in a concrete form what had been his physiological standard during life. There was in him a physiological barrier to his being a large feeder or a big drinker. We all know such persons—persons who cannot eat big dinners daily, or take alcoholic liquor of any kind regularly save in small quantity. The barrier is really a physiological one; the organs concerned are not capable of dealing with the amount or kind of work thrown upon them, and auto-intoxication or toxæmia of one kind or another results.

CASE 67.—This case illustrates a type of symptom common in old people with thickened vessels, physical weakness, and the deteriorated brain, so frequently regarded as the inevitable result of senility.

Such persons from time to time are seized with restlessness, insomnia, mental excitement, which amounts, during the night, to a form of delirium. They get out of bed during the night and wander about the room if allowed to do so, or do strange and dangerous things, as lighting matches, and interfering with the fire. When an attack of this kind is on, I always find that their sclerosed arteries have undergone marked contraction. I had some time ago, in the male sick ward of Queensberry House, a very pretty example of this condition. The patient was a very old man, but usually quiet and inoffensive, sleeping well, and moving about during the day, giving no trouble to anyone. When his attack came on he was as I have described. The striking feature, beyond the manifestations I have mentioned, was a remarkable contraction of his arteries. The radials, which were always somewhat sclerosed, but of good size, with a fair volume of blood in them, become reduced by contraction to half their ordinary size or even less. The infallible remedy for these attacks in this old man was a dose of paraldehyde.

If this was given at night he slept quietly, and next morning his arteries had returned to their ordinary condition. With a few days of very low diet, and sometimes a second dose of paraldehyde, he returned to his ordinary condition. No other hypnotic or sedative had the effect upon his arteries that paraldehyde had, and no other had any beneficial effect upon his attacks. This drug does not have a corresponding effect upon all such cases. In other cases the hypertonus which leads to the insomnia and restlessness can be counteracted by means of laxatives, or by sulphonal, trional, veronal, or phenacetin, or by a combination of two of these. The explanation of arterial contraction causing this train of symptoms is to be found in the anæmia of an enfeebled and degenerated brain—the anæmia being due to the vessel constriction; and the symptoms disappear as the constriction is removed.

Case 68.—Mrs. M., æt. 72, who had been for years an inmate of Queensberry House. On Sunday, 14th October 1906, she had a gastric attack, with sickness and vomiting. Under treatment these symptoms disappeared, but on Tuesday she was mildly delirious, with delusions. On Thursday morning she had complete aphasia and right hemiplegia; but gave her left hand when asked, and in other ways showed she was conscious. She was seen shortly after the onset of these symptoms and was ordered erythrol tetranitrate. On Friday the dose was increased. She rapidly improved, and in the course of a few days regained the power of speech to a considerable extent, whilst the paralysis of the limbs as steadily passed off. Within a fortnight she was walking about as formerly and speaking distinctly.

This patient had another attack of delirium in April 1907, with slight motor enfeeblement on the right side, while the speech became inarticulate. As the result of our former experience of her, the bowels were at once freely cleared out, and she was given trional at night. She was less restless, although she only slept for two or three hours, but the paresis was less. She then got thirty minims of paraldehyde, after which she had a whole night's sleep, and the paresis was entirely gone, although the speech, while distinctly improved,

was still indistinct. The two following nights she slept soundly without a hypnotic.

As the outcome of observations on this patient, it was found that at short intervals she became restless, excited, and wakeful during the night. When the first trace of these symptoms appeared, she was given two grains of calomel and a small dose of castor oil at bedtime, with the result that she was quiet and composed the following day. In the month of June after this course had been followed for some time, I took a hæmomanometer observation on the forenoon of the evening she was to be given the usual dose, and found it 140 mm. Hg., and the pulse 72; the following forenoon it was 125 mm. Hg., and the pulse was 80 and larger.

On July 24th, 1907, when dressing in the morning, the right hand began to shake, and then the whole right side. She was at once undressed and put to bed. I saw her soon after, and found her in a dazed condition, and not able to answer the questions addressed to her. She evidently could not speak; the right arm and leg were paralysed, but both limbs were jerking rhythmically. There was no response on the right side to tickling the sole of that foot; but tickling the right caused as active a response on the left side as when the left sole was tickled. She was given quarter of a grain of erythrol tetranitrate every four hours. The next forenoon, as soon as I appeared at her bedside, she raised herself into a sitting posture, answered questions promptly, and gave me her right hand. The nurse informed me that the jerking of the limbs ceased soon after the first dose of the erythrol was given. Between the 25th and the 31st the manometer pressures seemed to be very erratic, ranging between 140 and 170 mm. Hg., but finally settled down to about 130.

This leads to the third group in which paralysis or paresis became permanent after having been preceded by "warning attacks."

GROUP III.—PERMANENT PARALYSIS OR PARESIS PRECEDED BY "WARNING ATTACKS."

CASE 69.—J. F., æt. 72, was seized, on 5th February 1907, with slight paresis of the right side, and of the right

side of the face, and slight impairment of speech. He completely recovered the power in the arm and leg, but speech continued to be a little blurred, and the facial paresis persisted. In June he had another attack of feebleness on the same side, with a corresponding increase of the difficulty in speaking. Even after this second attack he was able to be out of bed, could walk across his room, and could articulate well enough to be understood. Mentally he was alert and cheerful. On 6th September he had complete left hemiplegia and could not speak a word. In various ways it was seen that he was quite conscious. I admitted him to the Infirmary the same day, and kept him under observation for a fortnight. At the end of that time the leg had regained a little power of movement, and occasionally he spoke a word or two, but he could not answer simple questions. In this condition he is likely to remain. The hæmomanometer observations made on this patient are referred to in previous chapters, so I do not repeat them here.

CASE 70.—Mrs. C., æt. 77, when returning home in the afternoon of 8th February 1907, went into a shop with the purpose of making a purchase, but could not tell the shopman what she wanted. She, however, was able to point to the article wanted. I saw her the following day, and found that she had a very interesting type of aphasia, the details of which need not be entered upon here. I admitted her to my ward in the Infirmary, where her type of aphasia was demonstrated to a number of medical men. For our present purpose it is sufficient to say that beyond the speech difficulty there was no symptom. There was no paresis of the limbs or face. She improved considerably, but there remained a measure of word difficulty. After a time she moved about as she had previously done, and went out freely. On the morning of the 25th July 1907, she had got out of bed and dressed herself as usual, when she suddenly felt giddy, and as if she were to fall. She sat down on a chair, and was helped to bed. I saw her about an hour afterwards, and found that she had left hemiplegia, the left side of the face being also affected. She was quite sensible, and talked volubly, as was her custom. The word defect was not appreciably greater.

On stroking the left sole there was marked dorsiflexion of all the toes, while a similar proceeding on the right side caused plantar flexion of all the toes. There was no loss of sensibility. The pulse was 68, and the arterial pressure 185 mm. Hg. Some observations were made by means of the hæmomanometer applied in the usual way, and also by means of Oliver's hæmodynamometer applied to the temporal artery, which was thickened and lent itself to such observations. There was a difference of 20 mm. Hg. between the two. Some improvement took place in the leg in this patient, but beyond that there is nothing to record.

CASE 71.—Mr. H., æt. 57, was seen with Dr. Inkster. The history was that sixteen months before I saw him he was seized with complete aphasia, when in his office, while at the same time he could not write. Although he recovered sufficiently to return to business, he continued to have a difficulty about words and the names of persons. He also had occasionally difficulty in writing—he would write a name wrong, know that it was wrong, but could not put it right. His speech difficulty was also worse when he felt tired. On one occasion he had a definite feeling of weakness in the left arm and leg. I saw him after he had been carefully dieted, and had taken iodide of potassium for some time. The radial artery was thickened and felt segmented, the brachial felt hard and thick, and the hæmomanometer gave a pressure of 185 mm. Hg. He was well nourished with a somewhat florid complexion, was said to be a comparatively spare eater and temperate in his use of liquor. All his life he had suffered from a "costive habit."

CASE 72.—Mrs. C., æt 68, was admitted to Queensberry House, and I saw her on 10th June 1907. The history was that three years ago she had a fit and lost the power of speech and of the right hand. She developed delusions, and was sent to an asylum, but was discharged in six months. Now and again she loses power in the right arm and leg, but she thinks she was never so powerless as at present. She says that she was walking about last week with the help of a stick, and that she lost power in the end of the week. The grasp of the

right hand was feeble, there was weakness of the right leg, but she could draw it up and extend it as she lay in bed. On the left foot the toe and ankle reflexes were normal to stroking the sole, on the right foot there was slight dorsi-flexion of the big toe, and no movement of the other toes, while the ankle reflex was active. The knee jerk was better marked on the left side than on the right. On 13th June she had improved sufficiently to be able to stand; the sole reflex of the right foot was again active; the pulse was 80, and the hæmomanometer gave a pressure of 200 mm. Hg.

On 29th June she was again seized still more seriously to all appearance. She was dazed looking and speechless, and she could not move the right arm and leg. I ordered half a grain of erythrol, which was not repeated, as the patient was in such a state of apparent collapse that I was afraid to repeat it. In the evening she rallied, for when the nurse asked how she was feeling the prompt reply was "fine." The following morning when I saw her she was sitting up in bed taking breakfast. Speech was rambling, but as the day passed she became more sensible. Power gradually returned on the right side. She had kept well up to the end of September, when this note was written. She is a cheerful, contented woman, able to be out of bed daily, and to move about her room. There is no impairment of speech. She is one of the patients in whom the radial artery suggests nothing near the pressure of 200 obtained from the brachial.

GROUP IV.—FATAL CASES OF HEMIPLEGIA.

This group is confined to fatal cases and in connection with them I seek to draw attention to the tendency to regard such cases as due to cerebral hæmorrhage. This tendency extends to the kind of case illustrated in the preceding group. The tendency to diagnose hæmorrhage seems to me to have become much more widespread amongst practitioners than it used to be, and this can only, I presume, be attributed to their teachers, for the opportunities of checking diagnoses are not large in general practice. I begin the group with two cases which may be taken as typical of their kind, and follow them with other two cases which, from their

clinical manifestations, might have been held to be cases of hæmorrhage, did not the post-mortem examination confirm the contrary opinion.

Two Fatal Cases Illustrating the Ordinary View.

Case 73 was an old gentleman of 71 years whom I saw in consultation. He had been seized on the morning of the day I saw him with right hemiplegia and aphasia. This occurred when he was slowly convalescing from what appeared to have been an attack of influenza with bronchopneumonia, so that his general condition was low and his circulation poor. The loss of power was not accompanied by loss of consciousness. The pulse when I saw him was soft and the blood-pressure low. There was no albumin in the urine. The patient became worse in a few days, and died comatose. The ordinary view of such a case would be that it began with a hæmorrhage, small in size, but sufficient while destroying some fibres to press upon adjoining ones; and that this initial hæmorrhage was followed by a second and a larger one in a few days. There was no *post-mortem* examination, and so there was no opportunity of checking the ordinary clinical opinion in this instance. The symptoms, however, closely resembled those in cases in which there was softening but no hæmorrhage, as proved by post-mortem examination, and I hold that these cases guide us as to the nature of the lesion in such cases as this.

Case 74 was a man aged 28, admitted to the Royal Infirmary with left hemiplegia without loss of consciousness. He had been in the Infirmary some months previously, and was known to have advanced chronic kidney disease. His arteries were thick and hard. He was markedly uræmic, sometimes noisy and talkative, at other times semicomatose or drowsy, but he recognised the Ward Sister, and could give the names of relations admitted to see him. In three days his temperature suddenly shot up to 105° to 106°, he became comatose, and died within twenty-four hours. My interpretation of the phenomena was that on admission the blood supply to the motor strand on the right side had been obstructed, and as his vessels were markedly constricted I leant to the

view of cerebral vessel constriction to explain the arrest. The blood-pressure was 180° by Oliver's hæmomanometer, taken about twenty hours after treatment had been begun. There was no alleviation of his condition save that he became less noisy. The final coma with the great rise in temperature I attributed to hæmorrhage. This case in so far as the main cerebral symptoms were concerned—the hemiplegia without loss of consciousness ending in profound coma in a few days—closely corresponded with the main phenomena in Case 73. The post-mortem examination here showed a large fresh clot in the usual region, involving the internal capsule and adjoining structures; round the clot there was a distinct area of softening.

In this case the symptoms on admission were referred to uræmia and to the arrest of blood supply to the motor strand in the right hemisphere; the later symptoms were referred to hæmorrhage. In this case also the ordinary view is that an initial small hæmorrhage is followed by a second, larger and fatal one. The paralysis without loss of consciousness is, however, satisfactorily explained by a local arrest of blood supply so complete as to lead to rapid softening of the portion of brain affected, while the later loss of consciousness resulted from hæmorrhage taking place into the already softened area. The appearances at the post-mortem examination warrant this contention, for the blood clot was quite recent, while there was a distinct zone of softened brain tissue round it. A minor observation in Case 76 gives support to this contention. The possibility of such a sequence of events has not, so far as I know, been hitherto considered, it being assumed that such cases were cases of hæmorrhage from outset to finish.

Two Fatal Cases with Softening.

Case 75 was a woman, aged 75, who had been an inmate for many years of Queensberry House. When I saw her in the forenoon she had paralysis of the left side of the face and of the left arm, she could draw the left leg up a little when asked to do so. She was conscious. The pulse was 80, and the blood-pressure was 135° to 140°. The

paralysis had come on in the early hours of the morning, and had been preceded by great restlessness. The women who occupied the same room volunteered the information that she had been "strange in her mind" for a week past, that she "knew nothing," and was "quite vacant." I saw her first on the 11th of November, and that afternoon she was put on small doses of erythrol tetranitrate, which were continued for some days, but was stopped, as there was no diminution in the degree of paralysis. On the 17th her face was flushed, her temperature was 101°, and her radial vessels were relaxed. She had slight bronchitis, and during the day her breathing seems to have become oppressed, and she died quietly that same evening. The post-mortem examination showed a subcortical area of softening, about the circumference of a florin, in the upper part of the parietal lobe, posterior to the ascending parietal convolution, the grey matter over it being a mere film. There was also distinct although moderate softening, involving the internal capsule on the same side. There was no hæmorrhage. The cerebral veins were engorged, and there was a large amount of fluid left in the skull when the brain was removed. The kidneys were atrophied and granular.

In this case the possibility of a secondary hæmorrhage was suggested by its presenting a certain resemblance to Case 74 in the terminal phenomenon of a rise in temperature, and yet it was found to be one of pure softening, with such a brain œdema as is frequently found in old people with atrophied kidneys.

CASE 76.—Peter M., æt 74, was the victim of a harmless delusion. On the night of the 9th December 1906, he became restless, got out of bed, wandered about the room in which he slept, and when I saw him on the morning of the 10th he was full of delusions. I admitted him to my ward in the Infirmary that same day. He was kept in bed and was quite quiet, but had a fixed delusion that he had been assaulted. On the morning of 15th December he was found by the nurse to have lost the power of the right side and to be speechless. When I saw him at 11.30 a.m., he was lying on his back with the eyes open and all the appearance of

consciousness: when spoken to he made no reply and gave no sign of hearing what was said to him. The face was not paralysed, and when he yawned the lips moved symmetrically. The right arm was not moved, while he moved the left one freely. The right arm was somewhat rigid at the elbow and shoulder; the forearm was flexed, and was straightened with some difficulty. The fingers were not rigid. Sensibility to

FIG. 32.—Section of brain from Case 76, showing three areas of softening, *a*, *b*, *c*. In *c* there was a small recent hæmorrhage.

FIG. 33.—Another section of brain from Case 76, showing area of softening at *b*. Dr. Carnegie Dickson kindly made the tracings from which these photographs were taken.

pinching was absent or much diminished in the right arm, but present in the left. He did not move the legs, but when they were passively drawn up they remained in that position. When pinched he drew them up a little. The response to tickling the soles was very active on the left side, but absent on the right. There was no ankle clonus. Both knee jerks were active. He had passed urine and fæces in bed. There was no albumin. The pulse was between 70 and 80 and

slightly irregular, the vessel wall was thick, the hæmomanometer registered from 180°–190°. He was put on erythrol tetranitrate, and on the 20th iodide of potassium was also given. No marked change occurred in the symptoms; on the 21st it was noted that he gave his left hand slowly when asked to shake hands. The patient died on the 27th of acute pleurisy, with some patches of broncho-pneumonia in the lobe affected with the pleurisy. The brain on the left side showed two considerable areas of subcortical softening, into one of which there was a small recent hæmorrhage; and a considerable area of softening into, and in the neighbourhood of, the lenticular nucleus.

In this patient death was the result of a definite intercurrent acute disease, so that the opportunity was afforded of checking the diagnosis of cerebral softening.

Sections of the brain at two different levels are shown in the accompanying figures. The softened areas are shaded. In the posterior area marked *c* there was a recent hæmorrhage about the size of a hazel nut. This had undoubtedly taken place into an area already softened, and tends to strengthen my opinion that fatal hæmorrhages may not infrequently be due to a primary softening, so that even *post-mortem* appearances may be quite misleading unless the possibility mentioned is fully recognised.

GROUP V.—TWO SPECIAL CASES.

CASE 77.—*Temporary paresis accompanying paroxysms of angina pectoris.*—In this patient, Mrs. M., æt. 49, the leading feature was the recurrence at intervals of about four weeks of symptoms typical of angina pectoris, while the most striking phenomenon was the association of left hemiparesis with them. She had her first attack of angina pectoris seven years before I saw her. During that attack the left arm was paralysed, but it regained the greater part of the lost power in twenty-four hours; still, she asserts that it has never been as strong as the other one since then. The history given to me by her medical attendant, Dr. Blackstock of Eskbank, was that every few weeks she suffered from angina pectoris; that when this supervened she became hemiplegic; and that

when the anginous attacks were cured the hemiplegia passed off. This corresponded with the history I gathered by questioning the patient myself; but I further ascertained from her that even when moving about the house, if she took an anginous attack, her left arm became affected, and that she had often lost the use of the arm for from twenty minutes to an hour. During the two occasions she was under my care in the Infirmary she had slight weakness of the left arm and leg, and there was also a partial or complete loss of tactile sensibility in these limbs. It is not necessary to record here all the details of this case, for the important facts are those I have given, and it is their interpretation which specially concerns me at present. I was fortunate enough to see this patient in a slight anginous attack, and in it there was the usual hypertonic contraction of her arteries. The recurring and close association of the anginous attacks with recurring hemiplegia or brachial monoplegia had been so frequently noted, that it seemed to me there was no possible explanation of the association but by regarding the abeyance of motor power to be due to a temporary lessening or cutting off of the blood supply to the motor strand in the brain, the result of a spasm contraction of cerebral vessels; the cerebral vessel spasm accompanying the systemic vessel spasm of the anginous attack. In this case changes of the kind mentioned seem to me a necessity for the explanation of the clinical phenomena, and it would be most unfortunate if physiologists regarded such phenomena as having no bearing upon their special department of investigation. Measures directed to the improvement of the general vascular condition led to very satisfactory results.

CASE 78.—*Old-standing right hemiparesis with temporary attack of complete right hemiplegia and aphasia.*—This case, a man of 58 years, was a partial recovery from an old hemiplegia. He was able to walk about and look after himself in spite of a disabled right arm and leg. His speech was somewhat mumbling, although he could speak so as to be understood. I saw him one morning soon after he had so completely lost power in his disabled limbs that he could not move them, while he had so entirely lost the power of speech

that he could not utter an articulate sound. He was quite conscious, and evidently greatly annoyed at his inability to speak, or to get out of bed. His vessels were thick and contracted, and measures were at once taken for their relaxation. He had considerably improved by the following day, and two days later he had regained his former power both of locomotion and of speech. That the symptoms here were due to an interference with the blood supply of the damaged area in his brain is, I think, beyond question; that he would not so speedily have recovered had the condition been local thrombosis is equally beyond question; while the occurrence of vessel constriction readily explains the loss of power—the prompt relaxation explaining the speedy recovery.

GROUP VI.—CHEYNE-STOKES BREATHING WITH THICK ARTERIES.

This case is interesting, and, taken along with the other observations in this chapter, is instructive, although meanwhile it stands by itself.

CASE 79.—E. K., a man aged 70, had been for a short time in Queensberry House. It is unnecessary to dwell upon his condition before he came under particular observation. In the middle of May 1907 he was confined to bed with, it was stated, difficulty of breathing. He was given spirit of nitrous ether and stramonium tincture. For some days his condition was very critical, so bad that the nurse had been summoned several times as it was thought he was dead. I saw him on the 28th May, when he was said to have somewhat improved. His condition was, however, still serious: he had marked Cheyne-Stokes breathing, the period of apnœa lasting for thirty-five seconds, the period of breathing for thirty seconds. This rhythm was quite regular during the times I saw him. The pulse varied from 48 to 120 per minute, being very irregular both in force and time. On the 29th his pressure was about 200 mm. Hg., the radial artery was much thickened and hard. The heart dulness was in the nipple line, there were no murmurs; there was no albumin in the urine. The breathing continued the same as on the previous day. The pulse was

very irregular, there being a large beat followed by a varying number of small beats, as shown in the tracing (Fig. 34).

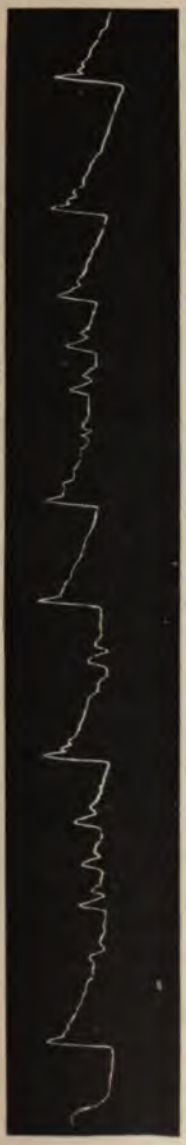

FIG. 34.—Tracing from Case 79, taken on 30th May. All waves present in radial artery, with haemomanometer pressure, 180 mm. Hg. ; at 190, small waves arrested ; at 200, all waves arrested.

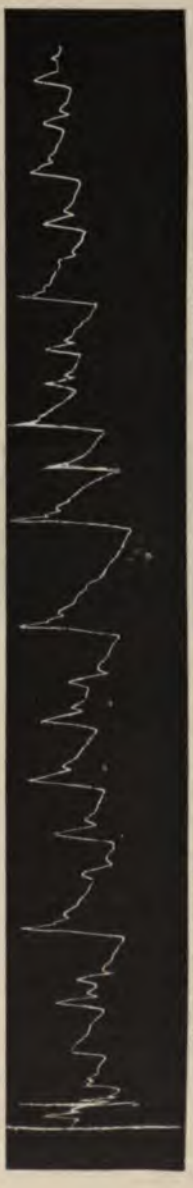

FIG. 35.—Tracing from Case 79, taken on 31st May, after treatment with erythrol, showing greater range of movement, the result of relaxation of arterial wall.

The observations by the hæmomanometer were specially instructive. I was using the wrist-pad to determine when the brachial artery became occluded, and I noted that with the pressure at 180 all the pulse waves were present in the radial; at 190 only the large waves seen in the tracing were present in the radial; while at 200 all waves were stopped. I put him on half a grain of erythrol tetranitrate four times in twenty-four hours and a mixture containing squill and iodide of potassium to be taken thrice daily. On May 31st I took the tracing (Fig. 35), the first half being taken during a period of apnœa, the second during a period of deep breathing. After a few days of this treatment great improvement was apparent. The Cheyne-Stokes breathing steadily lost its special character, he slept soundly during the night, and the pulse became much more regular. On June 9th the dose of erythrol was reduced

FIG. 36.—Tracing from Case 79, taken on 26th June when patient had recovered; hæmomanometer pressure, 200 mm. Hg.

to $\frac{1}{4}$ grain four times in twenty-four hours; and on June 10th it was stopped. The pulse was much more regular in force, and beat about 70 per minute. Notwithstanding the remarkable degree of improvement in this patient's condition, due undoubtedly to the erythrol, at no time did I get his pressure below 200 mm. Hg.; and yet that his capillary circulation was relaxed was seen by the rosy tint of his face and lips, and that the radial was somewhat softened was felt by the finger. I take it that the brachial was not appreciably influenced, and so the manometer reading remained much the same as before. On the 26th June I took the above tracing Fig. 36 from the radial artery. It shows the great improvement that took place in the pulse, representing, of course, a like improvement in the heart. The Cheyne-Stokes breathing had absolutely disappeared long before this.

The case was of varied interest, and I am not sure that I

ever saw so severe a case recover. The hæmomanometer observations were of special interest, as they so clearly showed that a rise of less than 10 mm. Hg. above 180° prevented the smaller waves, shown in the sphygmographic tracing, reaching the wrist; while a rise of less than 10 mm. more obliterated the large waves also.

Since the foregoing was written this man has had another seizure, but as soon as the symptoms appeared he was put on erythrol every four hours, and on iodide of potassium and squill thrice daily. He was kept exclusively on milk. He made a speedy recovery, and was able to be out of bed and to move about within a fortnight. The second attack was undoubtedly induced by the attentions of his friends in supplying a variety of additions to his ordinary plain dietary.

THE CLINICAL PATHOLOGY OF THE PRECEDING GROUPS OF CASES.

Transitory and recurring Phenomena.—It is unnecessary to multiply such cases, for if those given cannot be made the basis of a convincing argument, a larger number would not carry conviction.

The cases I have given seem to me to constitute a series of phenomena which are so linked as to form an unbroken chain; and which can only be satisfactorily explained and linked on the grounds which can now be indicated.

Minor Phenomena and Focal Spasm.—Beginning with the minor manifestations, such as those in Group I., I believe they are all explicable on the assumption that they were due to *anæmia of brain areas, that the local anæmia was due to hypertonic contraction or to spasm constriction of the vessels in those areas.* The spasm of cerebral vessels may have been an accompaniment of a general hypertonic contraction, and in some cases it undoubtedly was; that general contraction is a necessary accompaniment is by no means certain. The fact that local or limited arterial spasm has been recognised in migraine has been already referred to, also the fact that temporary local paralyses are known to accompany the migrainous attack. It seems to me that local spasm-con-

striction of cerebral arteries is made clinically certain by these observations. But this hypothesis can be supported by other clinical facts. I know a medical man who could not drink beer because it gave him migraine; and he had further observed that the temporal artery on the side of the migraine was hard and thick during the attack. Another medical man informed me that as a student he discovered that if he had poached eggs to breakfast he had an attack of migraine as a consequence. This same medical man told me that with him a migraine was usually accompanied by a certain difficulty in getting the right word. Without going further afield for evidence of the same kind, I hold that in such phenomena we have clear clinical proof that some substances (in one case a simple dietetic substance, in another what can ordinarily be regarded as a harmless beverage) act on certain persons as poisons, and confine their action to certain nervous areas, acting presumably on the blood vessels of the particular area affected. Very strong support is given to this view by the ocular phenomena which have been noted in severe cases of quinine poisoning. Again I am indebted to Mr. R. Marcus Gunn for information. He kindly sent me the following memorandum:—In severe cases of *quinine poisoning* the ocular symptoms are usually complete or partial blindness, associated with marked narrowing of the retinal arteries and pallor of the optic discs. Sometimes at first the ophthalmoscopic appearances resemble those present in embolism, or thrombosis, of the central artery. As a rule recovery takes place, but this is often much delayed. It is said that a second attack may be the result of a comparatively small dose. Pathologically, in lower animals, no changes have been found in the vessels during the first month or so, although they are ophthalmoscopically small. After two months, the arteries have been found to have thickened walls with contracted lumen.

Here, then, is a substance having, as part of its poisonous effect, a marked localised effect on the arteries, causing their constriction, and ultimately leading to the thickening of their walls and diminution in their lumen. It is difficult to see how we are to get evidence more conclusive than this. The vessels inside the brain can never be watched as the

retinal vessels can be watched; but I hold that my series of cases requires a similar clinical pathology for their interpretation, and few will hesitate to accept the interpretation as a valuable and practical working hypothesis in corresponding cases.

The Special Irritant.—The question of the special irritant, when there is merely focal vessel spasm in a brain region, can only, so far, be a matter of surmise. Still, even surmise may have a practically useful side. My hypothesis is that the spasm in such cases is caused by local fatigue products, or by waste products, so irritating the vessels as to cause their constriction. The effect of such products seems to me always to be of this nature. Experimentally it is known that fatigue products accumulating in muscle prevent its response to electrical stimulation. These products can be artificially washed out, after which the response to stimulation returns. It is, further, a well-known fact that hot bathing is one of the best means of recovering from a sense of fatigue. The action of the hot bath is so to dilate the vessels that the flow through them is facilitated and the fatigue products are more promptly removed. In migraine whatever relaxes the vessels cures the symptoms.

The Phenomena in Persons with Sclerosed Vessels.—There is another aspect of some of the cases with recurring cerebral phenomena which has to be dealt with. In persons with sclerosed or atheromatous arteries, my experience is that the cerebral phenomena are associated with hypertonic contraction of systemic vessels. I presume that in these cases the same cause which excites contraction of systemic vessels is operating on the cerebral vessels—that, in fact, there are substances in the blood which are acting directly on both, and are therefore not acting through the vasomotor centre. Of this association I am fully satisfied; the only point which appears to me to be open to question is whether this association is usually present in persons with normal vessels. My own experience does not allow me to form an opinion on this point. When hypertonic contraction is present in the systemic vessels, it gives great confidence in adopting the appropriate treatment, while the hæmomanometer provides, as a rule, a valuable record of the effect treatment is exercising on the vessels.

When, along with systemic hypertonic contraction, there is paralysis, it can be accounted for by the vessels in the particular area of brain affected having undergone greater structural alterations than elsewhere. This, however, leads now to the consideration of the cases of temporary and permanent paralysis in persons whose arteries are thickened.

Temporary and Permanent Paralysis in Persons with Sclerosed Vessels.

In Cases 68 and 77 I have shown how unreasonable, and even absurd, it would be to regard the recurring paretic phenomena as due either to hæmorrhage or to thrombosis. That multiple attacks of temporary paralysis could be caused by an equally numerous number of hæmorrhages is quite unthinkable, and this is also the position as regards thrombosis. The only explanation possible is to regard the paretic attacks as due to recurring partial arrest of the blood supply to the motor strand, and the only conceivable way in which this could be brought about is by vessel constriction. The systemic vessels showed this constriction, while cerebral vessel constriction occurring coincidently would explain the paretic phenomena.

In Case 78 the rapid recovery from the total paralysis seems to me to exclude the idea of either hæmorrhage or thrombosis having occurred in the already damaged motor strand in the brain. Either of these in such a damaged area would have inevitably led to a permanent loss of power, and yet no such loss resulted. I know of no other way to account for the temporary lowering of the circulation in the damaged area than by assuming that vessel constriction had occurred.

Looking further at Case 68, I again contend that it is most unreasonable to suppose that there was even a small hæmorrhage into the brain. A small hæmorrhage into the motor strand would destroy some fibres permanently, a fact equally true of thrombosis, unless the collateral circulation were very promptly established, which in the brain cannot be assumed as likely. The pronounced motor paralysis with aphasia must have been due to a partial arrest in the blood supply to the great motor strand; the arrest cannot have been complete, for had it been, softening would have taken

place rapidly, with a destruction of fibres from which there would have been no complete recovery. Here again the only satisfactory explanation of the complete restoration is found in assuming that there was cerebral vessel constriction diminishing blood supply for the time, but passing off, and again permitting the normal nutrition of the affected area. In this particular case the paralysis was preceded by a period of restlessness and mild delirium; such an upset of brain function as is to be attributed to a disturbance of cerebral circulation. The sequence of phenomena is fairly common in old people, and is noted in Case 76 also, and is an argument in support of my view that the paretic phenomena result from transitory vessel changes just as the mental phenomena do. Had the focal anæmia persisted or been complete, softening would assuredly have followed, from which there is no restoration. In this case treatment by a powerful vaso-dilator was early adopted, and, as has been seen, there was rapid recovery on three different occasions. The result can hardly have been mere coincidence, that is to say, that the patient would have recovered as rapidly had no such drug been used.

In Case 65 such marked improvement followed when treatment produced freedom in the capillary circulation, that it led one to attribute the improvement in the paretic phenomena to a like flushing of cerebral vessels.

In Cases 75 and 76 the softening in the internal capsule or its neighbourhood was so much less complete than in the cortical softened areas, that the possibility of the difference being the result of the administration of the same drug suggested itself. If any value is to be attached to the observations they would, of course, support the view of vessel constriction.

Cerebral vessel constriction, if it leads to complete arrest of blood supply to any portion of brain, will inevitably lead to softening of the area so deprived of nutriment. This diminution of blood supply to a local area may be due to spasm constriction of the vessels in the particular area; but it may also be explained in another way. If constriction of all the cerebral vessels occurred at the same time, those areas would suffer most where the lumen of vessels was per-

manently diminished by arteriosclerosis, by atheroma, or by obliterating endarteritis. These changes frequently assume such proportions that a general vessel constriction in the brain would deprive the areas, where vessels were so affected, entirely of their blood supply, and this would inevitably lead to softening. The three pathological changes mentioned cause diminution of lumen, and any general constriction of brain vessels would markedly diminish, and might even completely arrest the circulation through the vessels so altered. In addition to this, the result in some of the vessels would certainly be thrombosis, leading to permanent cutting off of blood supply and inevitable softening. The possibility of vessel spasm-contraction leading to thrombosis in sclerosed vessels, or in the atheromatous parts of constricted vessels, is a new view, and yet it naturally suggests itself when constriction is pictured as occurring in vessels which have undergone the permanent anatomical changes mentioned. Realising this, the occurrence of softening, even after relaxation has been induced, is readily explained, on the reasonable assumption that constriction had lasted long enough to permit of thrombosis. It is not necessary to argue that the conditions formulated are favourable to thrombosis occurring, for they correspond to those generally recognised as sufficient to produce that condition.

Softening or Hæmorrhage?

A further point to be considered is the relationship between softening and hæmorrhage, this being the question raised by the phenomena in Cases 73 and 74. In Case 74 we have both clinical history and post-mortem examination to help us to a conclusion. When this patient was admitted to hospital the mental unrest and delirium was uræmic in origin, but was not intense enough to prevent the patient recognising people and answering questions when directly spoken to—he was not unconscious, and yet he was hemiplegic. Active measures were used for the alleviation of the uræmia, the reduction of blood-pressure, and the relaxation of his vessels, the result being that he became quieter. In the course of three days the temperature

abruptly rose, and the patient became comatose, and died with symptoms of general brain compression. The ordinary explanation in such cases is that the hemiplegia without loss of consciousness is due to a small hæmorrhage, while the later coma is due to a second and larger bleeding. Chronic kidney disease with marked vessel changes, and high blood-pressure are common precursors of such cerebral phenomena, and are commonly regarded as sufficient to establish the diagnosis of hæmorrhage. There is, however, another explanation which to my mind is more satisfactory in my case. It is this, namely, that the hemiplegia without loss of consciousness was due to a focal anæmia determined by vessel constriction, and this led to thrombosis, followed by softening. Into this softened region hæmorrhage occurred in about three days, and of this the patient died. There is no doubt that the hæmorrhage was a terminal occurrence in this case, while the fact that it showed a zone of definite brain softening round it suggests the interpretation that the hæmorrhage occurred in what was primarily an area of softening. I am aware that softening around a hæmorrhage may be secondary, but the hæmorrhage in this case was too recent to have led to the softening. The facts in this case, and the sequence in which I place them might be accepted as also applicable to Case 73, where there was no post-mortem examination, were it not that Case 75 showed a somewhat similar sequence of clinical phenomena, and yet there was no hæmorrhage, only softening, to explain the hemiplegia: while the final condition, instead of being due to hæmorrhage, was due to venous engorgement and brain œdema, a common sequence in old people who become comatose and have atrophied kidneys with weak hearts.

Symptoms due to a Feeble Circulation.

The cerebral manifestations dealt with cannot be left without referring to another aspect of their clinical pathology. It is a not uncommon experience to see cases where the patient has found himself to be paralysed on awaking from sleep; or the nurse or a relative makes the discovery. The clinical pathology of such cases is, I believe, as follows,—during sleep cardiac

action is at its feeblest and the cerebral vessels are contracted. If the heart at its best be feeble, and if the cerebral vessels are sclerosed or atheromatous, the conditions during sleep are just those which would favour a focal anæmia which might be complete enough to lead to softening. That the motor tract suffers so commonly is, I imagine, due to its functional activity and to its vessels being most altered. In two of my cases the first areas softened were cortical areas, followed by softening in the great motor tract. These cases are of great importance from the standpoint of prevention, for I have seen cases where it appeared to me that fatal results have followed upon a vigorously applied reducing line of treatment.

Treatment.

The practical value of the foregoing contentions is that they explain and link together a great number and variety of clinical phenomena, the nature of which has been obscure; and that they provide a new point of view from which treatment can be considered. The autonomy of the cerebral circulation no doubt introduces a factor which theoretically presents a difficulty, for it has to be assumed that the condition of the systemic may not represent that of the cerebral circulation. In this connection, however, the fact that the condition or composition of the blood affects the measure of vessel contraction or tone has to be given a more prominent place than it has hitherto been accorded. I have submitted a number of clinical observations to show that cerebral symptoms coincided with systemic vessel constriction, and that the former disappeared when systemic relaxation was induced. At the same time, when the autonomous aspect of the cerebral circulation is regarded, it has to be acknowledged that it would probably be of much therapeutic value to have more experimental data than we possess. Meanwhile we have to be content with such facts as I have indicated in the earlier part of this chapter, adding, however, to these the results of clinical experience with the therapeutic measures we have at our disposal.

Of the considerable array of vaso-dilators the one

which usually acts most definitely and whose action can be most readily maintained is erythrol tetranitrate. The use of this powerful dilator has, however, often to be supported by the administration of digitalis, squill, or strophanthus. This is necessary when the condition of the heart is unsatisfactory as regards strength. The estimate of the blood-pressure within the constricted vessels becomes thus of first-rate importance. For instance, if a case of focal anæmia be mistaken for a hæmorrhage and reducing measures used, as is not uncommon, these may only slightly relax vessels, while, if the heart be feeble, they lower blood-pressure so much that complete focal bloodlessness may be induced with its irreparable softening and possibly a secondary hæmorrhage. If the constriction has already led to thrombosis, even prompt relaxation will not prevent a measure of softening with its corresponding permanent disablement. This cannot be always avoided, but the line of treatment indicated promises success in a considerable proportion of cases. Among the dilators which I have used may be mentioned spirit of nitrous ether, iodide of potassium, belladonna and its alkaloid atropine, stramonium, valerian, phenacetin, trional, paraldehyde, or combinations of these. There seems to me to be no doubt that when the circulation is looked at from the standpoint I have indicated, much can be done to prevent the occurrence of the changes referred to. When the systemic circulation as judged of by the finger and the hæmomanometer shows a ready response to treatment, the outlook is very favourable; but when cerebral symptoms occur without corresponding indications in the systemic system, the particular measures to be adopted are determined by the general estimate formed of the patient's vessels and heart. Measures can be directed to allaying the contractile irritability of the cerebral vessels while maintaining as large a volume of blood from the heart as possible. In no other department of practical medicine can so much be done for the prolongation of life, sometimes of great public value, and for the protecting of people from the sadness or misery of partial cerebral disablement, as can be accomplished in such cases by a right understanding of circulatory phenomena: and I am convinced that this understanding has at its foundation the recognition of hypertonic contraction of vessels; and

the separation of this condition from what is called blood-pressure. The hypertonus may be present when the hæmomanometer registers 115°, and it may be absent when it registers 200°. Sclerosed vessels and atheromatous vessels as such are not incompatible with long life, but as soon as the factor of hypertonic contraction comes in there is danger. Here, as in angina pectoris, the sensitiveness of the vessels can be reduced by rigidly regulating the diet on the lines I have already frequently indicated.

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CHAPTER XVII

CONCLUSION

It seems to me to be desirable as a conclusion to this study of arteries and of blood-pressure to join together the various links into such a chain as they form in my mind. The value or strength of the individual links will be differently appraised by different minds; and some of the links will doubtless yet be strengthened. I have, however, no doubt they form a chain sufficiently strong to serve as a safe and useful clinical guide to those not strongly committed to other views.

To myself the links naturally assume a chronological order,—chronological as regards their evolution in my own mind, while such an order does not appear to be in any considerable sense disadvantageous.

The first link in the chain is the clinical assurance that the tightening up and relaxation of the radial artery is a fact of great clinical significance. The second is the correction of the common conception that pressure is heightened inside all hypertonically contracted vessels.

It is indeed imperatively necessary unequivocally to separate the two factors of the vessel wall and the contained blood. The state of the vessel wall need present no initial difficulty; it is either thickened or not thickened; if thickened, the degree necessarily varies, and varies within wide limits. The other point is the blood-pressure inside the vessel. This can only be arrived at by putting aside all preconceived ideas of what is *supposed* to happen, or *ought* to happen, when vessels are thickened. The question of pressure inside the artery has to be approached from the simple standpoint of observation made by the cultivated finger. That the finger can do this is testified to by generations of skilled clinicians taking

the pulse as the test of heart power, and if the test of heart power, necessarily of the blood-pressure inside the vessels. This being so, the controversies as to the meaning of the words *tension* and *elasticity* are merely of academic interest, for they do little or nothing to simplify the clinical position. The less involved the question can be made, the more likely is the individual judgment to be correct, and in a medical school the student and future practitioner has to be considered. To be able to estimate the pressure inside the artery, no matter what the thickness of its wall, is the lesson to be learned, and the learning of it is to be kept steadfastly in view. That it can be attained is, to my mind, unquestionable. It was never seriously questioned until the wrong interpretation of hæmomanometer readings was supposed to discredit the value of the sense of touch.

Thickness of the vessel wall is easy to recognise. The thickness, as it varies in the twenty-four hours, and in different conditions or diseases, is also soon recognised, when the fact of its occurrence is deemed worthy of attention. It will then be found that sclerosed vessels as well as normal vessels vary in thickness. The thickening will be found associated with all the causes which have been enumerated,—sometimes it is a nervous reflex, more commonly it is the result of blood impurity. It is a great step to recognise clinically that not only normal but sclerosed vessels tighten up and relax, for it at once takes away the conception of the rigid tube.

Having settled these points, the next point is to determine what is meant by the diffuse arterio-sclerosis, which was described as if it were atheroma. The solution of this was sought for in the radial artery, as it is the condition of that vessel during life which leads to the diagnosis of arterio-sclerosis. The changes found in the radial and other arteries have been described, and my contention was and is, that the condition is not only totally different from atheroma, but that the changes in themselves clearly indicate their true nature.

The continued irritative stimulation of the arterial wall, manifested clinically in the sustained or recurring hypertonic contraction, leads inevitably to such structural changes as

have been described. The fact that a fibrous hyperplasia of the tunica intima takes place, at once shows that the vessel changes are not to be attributed to a mere nerve influence, but to the blood itself. The intensity of the intimal changes inside the kidneys appear to me to clearly indicate the prolonged presence of excrementitious substances in the blood seeking an outlet by their usual channel. Here, in short, lies the solution of the problems as to renal, cardiac, and arterial changes. Long-continued blood conditions preceded the vascular, cardiac, and renal changes so well known. If the kidney itself was the seat of a primary inflammation of the subacute type, the blood condition necessarily resulting led to the widespread changes of arterio-sclerosis and heart hypertrophy. The development of heart hypertrophy as a result of the raising of aortic pressure has not appeared to me to require to be dealt with here.

After the recognition of these facts, the problem of blood-pressure, as it was estimated by the clinician and thought of by the physiologist, presented great difficulty. The sphygmograph, the arteriometer, the hæmodynamometer of Oliver, an occasional use of the Riva-Rocci sphygmometer, did not seem to me to help the solution of the problem. The advent of the modifications of the Riva-Rocci instrument led to a more sustained use of them, and the results when placed alongside the evidence obtained by the sense of touch seemed to be hopelessly conflicting. Amongst one's friends or colleagues the same conflicting evidence was available; some allowed the manometer to supplant their ancient faith in the sense of touch, others treated the evidence afforded by the instruments with a neglect bordering on contempt. The same diversity of view seemed to prevail widely. My solution of the problem has been presented in the preceding chapters. The condition of the arterial wall has been shown to be the added factor causing the high hæmomanometer readings obtained in pathological conditions. The state of the arterial wall, and the relation of its thickness to the size of the lumen, is determined not only by permanent structural thickening, but by the thickening of hypertonic contraction. The steps from hypertonus to sclerosis of vessels and arterio-sclerotic granular kidney have been indi-

cated; the causes of hypertonus have been examined, and it has been shown that the direct influence of the blood composition upon the vessel wall must have a predominant place given to it in this connection. Experimental data and all clinical experience are available in support of this generalisation.

I need not add to the length of this monograph by adding a special chapter on treatment. In various of the preceding chapters I have dealt with treatment and prevention sufficiently to indicate the lines I follow. I shall only repeat that the aim is to prevent hypertonic contraction. If the vessels are already sclerosed, the prevention of hypertonus will often prevent the loss of the faculties the possession of which makes life worth living. To those who are well on in life and do not enjoy the readings of the hæmomanometer, there is abundant solace available in the abandonment of the idea that blood-pressure rises with advancing life. Even if arteries are permanently thick, life need not be materially shortened if hypertonus be prevented, and if steps be taken to allay arterial irritability; and these are undoubtedly within our control. If hypertonic contraction be avoided there need be no fear of cerebral hæmorrhage as life advances. The changing tonus of the thickened vessels can be followed by the hæmomanometer, and the records correctly interpreted are a valuable guide to prophylaxis. The instrument ought, however, to be called the **Angiomanometer** when used as an instrument in clinical pathology, for the clinician must recollect that, given thickened vessels, the instrument records not blood-pressure, but **arterial resistance.**

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